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THE EFFECTS OF THREE DIFFERENT POSITIONS ON ARTERIAL OXYGEN AND RELATIVE PULMONARY SHUNT IN POST-OPERATIVE CORONARY ARTERIAL BYPASS GRAFT PATIENTS

BY

MONICA CHAN ©

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND
RESEARCH IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR
THE DEGREE OF MASTER OF NURSING

FACULTY OF NURSING

EDMONTON, ALBERTA

(SPRING, 1991)

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THE EFFECTS OF THREE DIFFERENT POSITIONS ON ARTERIAL OXYGEN AND RELATIVE PULMONARY SHUNT IN POST-OPERATIVE CORONARY ARTERIAL BYPASS GRAFT PATIENTS

SUBMITTED BY MONICA CHAN

IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF MASTER OF NURSING

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ABSTRACT

Positioning is an integral part of nursing care for the critically ill patient. Routine turning of patients from lateral positions to the supine position is carried out without consideration to the underlying pulmonary pathology. In coronary artery bypass graft (CABG) patients, the incidence of left lower lobe atelectasis is very high. Positioning such patients in the left lateral position may create a situation of ventilation/perfusion mismatch. This mismatching may lead to hypoxemia as a result of pulmonary shunt. The purpose of this study was to examine the effects of three different positions (30° head of the bed elevated and supine, 30° head of the bed elevated and left lateral position, 30° head of the bed elevated and right lateral position) on arterial oxgyen and relative pulmonary shunt in mechanically ventilated post-operative CABG patients.

A repeated measures design was utilized on a convenience sample of 30 post-operative CABG patients. Ninety percent of the subjects had post-operative atelectasis. The findings of this study showed no significant differences in arterial oxgyen and relative shunt among the three positions. The results indicate that positioning may not be an important consideration in arterial oxygenation in post-operative CABG patients, who are mechanically ventilated with a high tidal volume-low frequency and positive end-expiratory pressure.

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CHAPTER I

Introduction

All things on earth, including human beings, are subjected to gravitational forces. The influence of gravity plays an intricate part in everyone's life. In healthy individuals, the effects of gravity may not be obvious because of the body's efficient compensatory mechanisms. In illness, the adaptability of the body is reduced; changes in posture may result in abnormal body responses. Intuitively, nursing has incorporated the importance of gravity in various aspects of nursing practice to promote optimal bodily functions. However, the effects of positioning on physiological functions have not been adequately investigated; thus the importance of this simple nursing intervention is undervalued.

The aim of most intensive care units (ICU) is maintenance of homeostasis of critically ill patients; positioning has a vital role in respiratory homeostasis. ICU patients are often in a supine position for long durations, as this position is considered most suitable for monitoring hemodynamic parameters and providing care. If patients are repositioned, it is done without consideration to any underlying lung pathology. In patients without pulmonary abnormalities, the different positions do not significantly affect gas exchange (Zack, Pontoppidan, & Kazemi, 1974; Remolina, Khan, Santiago, & Edelman, 1981). But pulmonary complications are common among hospitalized patients, especially critically ill patients (Grosmarie, 1983; Tyler, 1984). One of the commonest complications among post-operative coronary artery bypass graft (CABG) patients is left lower lobe (LLL) atelectasis (Good, Wolz, Anderson, Dreisin, & Petty, 1979; Chulay, Brown, & Summer, 1982; Jansen & McFadden, 1986; Weiland & Walker, 1986; Wilcox et al., 1988; Chin, Modry, & King, 1986; Matthay & Wiener-Kronish, 1989). Several studies (Zack et al., 1974; Remolina et al., 1981; Piehl & Brown; 1976) have demonstrated improvement in oxygenation when the patient's position is adjusted to compensate for lung pathology. It seems logical to utilize positioning as a nursing intervention to improve gas exchange in post-CABG patients. However, there are inconclusive reports of the effects of patient's positioning on pulmonary gas exchange in the presence of atelectasis. Banasik, Bruya, Steadman, and Demand (1987) found that the arterial partial pressure oxygen (PaO_2) was higher in the right lateral and supine positions than in the left lateral position of post-CABG patients. The authors suggest that the positional improvement in oxygenation is due to better ventilation/perfusion ($\dot{V}/\dot{Q}$) matching. Ventilation is the process of getting air into and out of the alveoli, whereas perfusion is the process of blood flowing through these alveoli for gas exchange. Improved oxygenation with better $\dot{V}/\dot{Q}$ matching is supported by other researchers who studied unilateral lung disease and oxygenation (Zack et al., 1974; Dhainaut, Bons, Bricard, & Monsallier, 1980; Remolina et al., 1981; Sonnenblick, Melzer, & Rosin, 1983). In a recent study, Shively (1988) found that there were no differences in oxygenation among CABG patients assuming the three different positions (supine, right lateral, left lateral). A major fault in Shively's study was the erroneous use of mixed venous oxygen saturation (SvO_2) as an indicator of arterial oxygenation. SvO_2 is an indicator of tissue oxygen consumption and not a true measure of arterial oxygenation (Ahrens, 1990). Shively's study thus neither supports nor disproves the study by Banasik et al. (1987).

Purpose of the Study

The purpose of this study is to clarify the effects of different positioning on pulmonary gas exchange in mechanically ventilated post-operative CABG patients. It is important for post-operative CABG patients to have adequate oxygenation because hypoxemia may cause tachycardia, tachypnea, and increased myocardial oxygen consumption. The sequelae of these changes may include cardiac arrhythmias, possible myocardial infarction, coronary artery graft occlusion and prolong ICU stay. This study of the effects of the three different positions (supine with head of the bed (HOB) elevated to 30°, left lateral with HOB elevated to 30°, right lateral with HOB elevated to 30°) on oxygenation will provide valuable information as to whether positioning is an important consideration for efficient oxygenation in mechanically ventilated CABG patients.

Significance of the Study

The results of the study may assist nurses to better understand the importance of positioning in ventilated post-operative CABG patients. The findings of this study may provide the rationale that routine turning of post-operative CABG patients from side to side may not be beneficial. Each patient needs to be evaluated individually as to which position will maximize the $\dot{V}/\dot{Q}$ ratio and consequently improve the PaO_2. In addition, the findings of this study will add to the basic scientific understanding of how position changes are related to pulmonary function in this group of patients.

CHAPTER II

Literature Review

Oxygenation and relative pulmonary shunt are affected by multiple factors. The purpose of this review is to look at some of the factors that might contribute to worsening oxygenation and shunting in post-operative CABG patients. An overview of previous studies on positioning and oxygenation will be presented. In order to appreciate the premise of this study, a basic review of pulmonary physiology is included.

Positioning and Pulmonary Physiology

The fundamental function of the lung is gas exchange. Gas exchange involves coordinating the process of ventilation and perfusion. These processes are influenced by gravity; therefore body positioning is an important consideration in pulmonary physiology.

Ventilation

Normal pulmonary ventilation is unevenly distributed in the lungs (West, 1977, 1978, 1985). The anatomical difference between the lungs creates a bias in which the right lung is always slightly better ventilated than the left (Nunn, 1987). Spatial differences also contribute to uneven ventilation. The seminal work by West (1977) demonstrated that preferential ventilation occurs in the dependent regions of the lung regardless of body positioning. West explains this differential ventilation is related to the weight of the lung, and weight is influenced by gravity. Encasing the lung is the chest wall and the diaphragm. In an upright position, the weight of the lung is mostly at the base and a higher pressure at the base is required to support this weight so that the lung will not herniate to the abdominal cavity. The pressure around the base of the lung is therefore higher than the pressure around the apex of the lung. The difference in intrapleural pressure within the thorax implies that each region of the lung operates at a different portion of the pressure-volume curve. The apical region of the lung is located near the top of this curve, whereas the basal region of the lung is near the bottom of this curve. On inspiration, the base of the lung will inflate more than the apex. The reason is that the apical region has a larger resting volume so a higher expanding pressure is required for inflation.

Obviously, ventilation is affected by lung volume. During shallow breathing at low functional residual capacity, the airways and the alveoli in the base of the lung are small, the intrapleural pressure at the base exceeds the airway pressure, thus promoting airway closure. In this situation, gas distribution is directed mostly to the apex of the lung. In contrast, during deep breathing, the intrapleural pressure becomes more negative and ventilation is preferentially directed to the base of the lung. Differing levels of inspiratory lung volume will shift the location of the apical and basal lung regions along the pressure-volume curve and change the compliance of these regions.

Perfusion

Adequate ventilation does not guarantee effective gas exchange. In mechanically ventilated animals in which a cardiac arrest was induced, in spite of adequate ventilation, the arterial partial pressure oxygen (PaO_2) and arterial partial pressure carbon dioxide ($PaCO_2$) levels were incompatible with life (Nunn, 1987). Therefore, gas exchange is also dependent on blood flow through the pulmonary capillaries. The major determinants of pulmonary perfusion are cardiac output, pulmonary vascular resistance, and body positioning (Philbin, Sullivan, Bowman, Malm, & Papper, 1970; Robichaud, 1990; Ng & McCormick, 1982; Reischman, 1988).

The pulmonary circulation is a low-pressure system with limited ability for selective perfusion; blood flow is primarily governed by gravitational forces (West, 1977; Dantzker, 1987; Nunn, 1987). Pulmonary perfusion is dependent on body positioning, with preferential blood flow to the dependent regions of the lung (Nunn, 1987; West, 1977; Ng & McCormick, 1982; Kaneko, Milic-Emili, Dolovich, Dawson, & Bates, 1966; Bhuyan, Peters, Gordon, Davies, & Helms, 1989). West (1977) explains that this pattern of blood dispersion is from the effect of hydrostatic pressure on the blood column and the inter-relationship of alveolar, pulmonary artery, and pulmonary venous pressures. Hydrostatic pressure increases down the lung, and so does the blood flow; there is thus a recruitment of previously collapsed vessels. In addition, at the bottom of the lung, pulmonary arterial and venous pressure exceed alveolar pressure; flow is determined by the arterial-venous pressure differences, as flow increases the vascular resistance decreases, explaining the preferential perfusion to dependent regions of the lung in various body postures. The three distinct zones (1, 2, 3) of blood flow which West describes are the results of the interactions of those variables.

Lung volume also influences the distribution of blood flow. During deep inspiration, the extra-alveolar vessels are pulled open by an increase in negative intrapleural pressure, subsequently diminishing the vascular resistance in these vessels and increasing the flow, especially to the dependent regions (West, 1977). On the other hand the exact opposite is observed with shallow breathing. At low lung volumes, the intrapleural pressure becomes positive at the dependent regions and causes compression of the extra-alveolar vessels. The repercussions are raising vascular resistance and compromising blood flow to the dependent areas. In other words, at high lung volume breathing, preferential perfusion occurs to the dependent region of the lung. At low lung volume breathing, blood is diverted to the non-dependent areas.

Ventilation/Perfusion ($\dot{V}/\dot{Q}$)

Generally, blood flow and ventilation increase linearly to the dependent regions of the lung, but the rate of change in blood flow is greater than that of ventilation (West, 1977; Nunn, 1987). The different rate of change in ventilation/perfusion results in the basilar alveoli being over-perfused relative to their ventilation and the apical alveoli being under-perfused relative to their ventilation. Therefore, variable $\dot{V}/\dot{Q}$ inequality exists within the lung, with a high $\dot{V}/\dot{Q}$ ratio at apex and a low $\dot{V}/\dot{Q}$ ratio at the base (West, 1977). The PaO_2 value is the summation of the different $\dot{V}/\dot{Q}$ ratios.

Positioning and Lung Volumes

Many researchers report that the major cause in alteration of lung volumes in different postures is because of the effect of gravity on the diaphragm and abdominal structures (Crosbie & Myles, 1985; Baydur, Behrakis, Zin, Jaeger, Weiner, & Milic-Emili, 1987; Jenkins, Soutar, & Moxham; 1988; Hough, 1984; Crosbie & Sim; 1986). The importance of lung volume change is its effect on functional residual capacity (FRC). FRC is the volume of gas left in the lung after normal expiration, and this is composed of residual volume and expiratory reserve volume (West, 1985). Studies of healthy subjects using the Helium dilution technique demonstrated that FRC is highest in an upright position and progressively declines with recumbency (Nunn, 1987; Jenkins et al., 1988). However, Jenkins et al. noted that FRC reduction is significantly less in lateral positions than in supine.

Hough (1984) explains the differences in FRC with positioning. The diaphragm is the major inspiratory muscle; factors which alter diaphragm excursion will alter lung volume. Keeping this in mind, when one assumes a supine position, gravitational forces dictate that the greatest pressure is exerted on the posterior regions of the dome-shaped diaphragm. In fact, Hough noted a 10 cm displacement of the diaphragm between sitting and supine positions. In lateral positions, the lower dome of the diaphragm is pushed high into the chest by the compressive forces of the abdominal contents, and the upper dome is flattened; therefore, FRC reduction is less dramatic than when supine.

Changes in lung volumes may not be significant in young healthy individuals, but in aged and ill individuals changes in FRC from different headrest positions have important ramifications for the development of pulmonary complications. The importance of FRC is its relationship to the closing capacity (CC). CC is comprised of residual volume and closing volume (CV) and is normally less than FRC. If FRC is lower than CC, then airway closure will occur. Aging causes a decrease in the elastic recoil of the lung, a decrease in the oxygen diffusing capacity, and an increase in the chest wall rigidity (Nunn, 1987). Because of the alterations in elasticity of the lung and the expendability of the chest wall, the intrapleural pressure is higher at any given lung volume, and the CC is higher for the elderly. Rea, Withy, Seelye, and Harris (1977) confirm in their study that the CV is age-dependent and that FRC is position-dependent. Nunn (1987) cites a study which examined the relationship between age, position, CC, and FRC, with the following observations being made: at mean age of 44 years, the CC equals FRC in the supine position; at the age of 66 years, the CC equals FRC in an upright position. Accordingly, in an elderly person in the supine position, CC will likely be higher than FRC in the dependent regions of the lung, leading to airway closure in the dependent areas of the lung and creating $\dot{V}/\dot{Q}$ inequality. However, a reduction in FRC secondary to cephalic displacement of the diaphragm is partially compensated for by enhanced contractility of the over-stretched dome-shaped diaphragm. Hence, during deep breathing, gas distribution to the dependent lung is about twice that of the upper lung (Nunn, 1987), indicating that the dependent and non-dependent lung are operating in different portions of the pressure-volume curve. Therefore, the $\dot{V}/\dot{Q}$ ratio of each lung remains relatively constant regardless of positions.

Influential Factors for Atelectasis Formation

The incidence of atelectasis in post-operative CABG surgery is high. This may increase the morbidity and mortality for this group of patients during the post-CABG period; hence, it is important to look at the different factors that might contribute to alteration in gas exchange seen in the post-operative period.

Effect of Anaesthesia on Pulmonary Function

Coronary artery disease (CAD) is exclusively a disease of the aging population (Christakis et al., 1989). The current medical, surgical or combination therapy is aimed at symptomatic relief. Coronary artery bypass graft is the surgical intervention for CAD. The surgery requires a long anaesthetic time and cardiopulmonary bypass, predisposing an individual to pulmonary complications--especially atelectasis--in the post-operative period (Chin et al., 1986; Myrer, 1986; Matthay & Wiener-Kronish, 1989).

An anaesthetic agent mediates its action through the central nervous system and is known to alter lung mechanics, $\dot{V}/\dot{Q}$ distribution, and gas exchange (Matthay & Wiener-Kronish, 1989). Atelectasis and hypoxemia are common following anaesthesia (Hedenstierna, Tokics, Strandberg, Lundquist, & Brismar, 1986). Using the computerized tomography (CT) scan in eight healthy patients to study the development of atelectasis during anaesthesia, Hedenstierna et al. reported shunting and dense areas in the dependent regions of the lung after 15 minutes of anaesthesia. A significant positive correlation between atelectasis and shunt, and a negative correlation between atelectasis and PaO_2, were noted.

The formation of atelectasis during anaesthesia is related to reduction in FRC (Schwieger, Gamulin, & Suter, 1989). During anaesthesia and mechanical ventilation in the lateral position, the volume of the dependent lung is reduced (Baehrendtz & Klingstedt, 1984; Larsson, Jonmarker, Lindahl, & Werner, 1989). This is due to the combined effect of the weight of mediastinal structures and the cephalic displacement of the relaxed diaphragm by the abdominal viscera (Baehrendtz & Klingstedt, 1984). Since diaphragmatic displacement reduces the area of the thorax and alters the lung volume, the result is a drop in FRC. Compounding the problem, during positive pressure ventilation the non-dependent regions of the lung ventilate better than the dependent regions of the lung (Robichaud, 1990; Krayer, Rehder, Vettermann, Didier, & Ritman, 1989). The dependent area has a higher resistance for movement of the diaphragm than the non-dependent regions of the lung (Krayer et al., 1989). When the FRC is below the CC, small airways close in the dependent regions of the lung due to lower transpulmonary pressure (Baehrendtz & Klingstedt, 1984; Schwieger et al., 1989).

Small airway closure changes the lung compliance and gas distribution. Baehrendtz and Klingstedt (1984) compared conventional and differential ventilation during anaesthesia in both supine and lateral positions. Data from differential lung ventilation showed that the dependent lung received 22% less volume and had a higher peak airway pressure than the non-dependent lung. However, preferential blood flow to dependent regions was unchanged because of the effect of gravity and the increase in transmural pressure in the non-dependent alveoli caused by mechanical ventilation. The alteration in gas distribution is explained by a reduction in compliance and an elevation in resistance of the dependent lung. Preferential ventilation toward the non-dependent lung, combined with preferential perfusion of the dependent lung, resulted in $\dot{V}/\dot{Q}$ mismatch. In the supine posture, a similar $\dot{V}/\dot{Q}$ mismatch occurred (perfusion toward dorsal and ventilation toward ventral regions), but the effect was less pronounced because of the short vertical distance and the lesser compression of the mediastinal organs in the dependent lung regions. This $\dot{V}/\dot{Q}$ abnormality is manifested as a widened alveolar to arterial partial pressure oxygen difference ($P(A-a)DO_2$), an increased venous admixture, and a lower PaO_2. Normally, hypoxic pulmonary vasoconstrictor response (HPV) will minimize the impact of these changes on gas exchange, but this response is blunted during anaesthesia. Inhibition of the HPV response will worsen oxygenation because of the development of absorption atelectasis in the lower lung region. Adding to the problem, bronchial secretions tend to gravitate toward the dependent lungs, enhancing the formation of atelectasis.

The influence of anaesthetic is carried over in the post-operative period. Frequently, in this period, further deterioration in gas exchange is seen. This is because of a further reduction in

FRC secondary to an increase elastic recoil of the lung by anaesthesia, a change in the activity of the respiratory muscles by the surgical incision, and the continual presence of interstitial edema and small airway closure which occurs during the surgery (Schwieger et al., 1989). On the whole, the action of the anaesthetic agent causes a decrease in FRC, alters the normal HPV response and promotes atelectasis formation. These effects may last for some time after the adminstration of the agent has ceased.

Effect of Cardiopulmonary Bypass on Pulmonary Function

The success of cardiac surgery is made possible with the advent of the cardiopulmonary bypass (CPB) in the 1950's (Callaghan & Wartak, 1986; Murkin, 1989). CPB is the procedure in which the functions of the lungs and the heart are carried out by artificial means of the CPB machine, which requires systemic hemodilution, hypothermia, and anticoagulation (Weiland & Walker, 1986; Walker & Weiland, 1986). This renders a quiet and dry field for the surgeon to perform the surgery and at the same time maintains tissue oxygenation and perfusion. Although most patients tolerate being on CPB well, a certain degree of multisystem dysfunctions following CPB are unavoidable. Some refer to these derangements as "post pump syndrome" (Matthay & Wiener-Kronish, 1989). Complement activation during CPB is believed to be the major mechanism for this syndrome (Weiland & Walker, 1986; Matthy & Wiener-Kronish, 1989).

It is a general belief that there is a relationship between the duration of CPB and post-operative pulmonary problems. The premise for this belief is that pulmonary ventilation is halted and perfusion of the pulmonary capillaries bed ceases during CPB (Weiland & Walker, 1986). When the alveoli are not distended, and this is combined with inadequate perfusion to the alveolar epithelium, the result is an insufficient or abnormal production and release of surfactant from type II alveolar cells (Weiland & Walker, 1986; Matthay & Wiener-Kronish, 1989). The abnormalities of surfactant production and function may be further accentuated by exposure of the lungs to the atmosphere and the cold temperature during surgery. Abnormalities in pulmonary surfactant may lead to an increase in secretion retention and result in collapsed alveoli (Matthay & Wiener-Kronish, 1989).

Another mechanism that may increase atelectasis is the passive diffusion of cold cardioplegia into the pulmonary micro-circulation during CPB when the right atrial sump is not used (Matthay & Wiener-Kronish, 1989). The high potassium concentration of the cardioplegia is toxic to the capillary endothelium or to the alveolar epithelium, which could predispose abnormal surfactant synthesis and function (Callaghan & Wartak, 1986). However, in a recent study, the findings contraindicated this general belief about cardioplegia. Lindberg, Ovrum, Abdelnoor, Vatne, and Holen (1989) studied two groups of patients--in one group of patients the right atrial drain was not used during CPB while the other group had the right atrial drain during CPB. Lindberg et al. compared the pulmonary gas exchange of these two groups of patients at different post-operative period. These researchers found no significant alteration in gas exchange and shunt, but the pulmonary vascular resistance index was significantly lowered when the cardioplegic solution was not drained. Their data during post-CABG indicated that shunting was stable for 24 hours, then a significant increase occurred afterwards. Radiographic changes were obvious immediately after surgery and progressively worsened for 48 hours before changes were stabilized. There were more changes on the left side than the right (Lindberg et al., 1989).

The changes in shunt and X-ray may be related to the sequelae of CPB. The sequelae of CPB are insufficient alveolar distention, decrease in lung compliance, alveolar collapse, retention of secretions, breakdown of capillary walls, and sequestration of blood in the microcirculation (Weiland & Walker, 1986). These effects may predispose microthrombi which may cause intrapulmonary shunting, interstitial edema, hypoxemia, and atelectasis.

The duration of CPB may have some relationship to pulmonary shunting. Bushman, Hall, Reves, Peterson, and Datkins (1985) studied 17 CABG patients with no known history of pulmonary abnormality and looked at shunt in three different time periods of CPB--pre-CPB, 10 minutes after CPB discontinued, and one hour after CPB. They reported CPB increases shunting but the cause is not CPB time-dependent. However, there are studies that have shown acute alveolar flooding and increased pulmonary capillary permeability in the post-operative period when CPB exceeds 150 minutes (Matthay & Wiener-Kronish, 1989). Of the patients that Bushman et al. (1985) studied, all but two subjects had CPB of less than 150 minutes.

Other possible causes for pulmonary embarrassment post-CPB have been attributed to a decreased red blood cell deformability (Hirayama, Roberts, Allers, Belboul, Al-Khaja, & William-Olsson, 1988), an activation of inflammatory mediators (Nilsson et al., 1988), a blood transfusion reaction and an idiosyncratic drug (Protamine) reaction (Matthay & Wiener-Kronish, 1989).

The severity of post-pump syndrome is a function of age. Boldt, Van Bormann, Kling, Mulch, and Hempelmann (1987) divided 40 CABG patients into two groups--under 65 and over 65 years old--to determine their response to CPB. They found that extravascular lung water (EVLW) was significantly higher in the older group post-CPB. No significant differences were noted between groups except a reduction in ejection fraction and PaO_2/FiO_2 ratio; an elevation in shunting and fluid balance were observed in the older group. When age and shunt were correlated with EVLW, a positive relationship was obtained. The increased EVLW in the older age group may be due to fluid shift secondary to alteration of capillary permeability and myocardial depression. The positive fluid balance without a concurrent increase in filling pressure might indicate fluid displacement into the interstitial space.

Other clinical reports show no significant increase in EVLW post-CABG surgery despite a 50% reduction in colloid osmotic pressure (COP) during CPB (Kowalski, Downs, Lye, & Oppenheimer, 1989). Hemodilution during CPB is primarily accountable for the drop in plasma COP. The reduction in COP could promote fluid shift into the interstitial space. On the contrary, Rein et al. (1988) found that reduction in plasma COP post-CPB was partly compensated for by a decline in interstitial COP with a concurrent rise in interstitial hydrostatic pressure. The lymphatic system functioned to drain excess fluid from the interstitial space, thus preventing edema formation. This delicate balance is often threatened with excessive fluid resuscitation in the early post-surgery period. Alternating the forces governing the fluid movement across the vessels will result in a net efflux of fluid from the vessel, and an increase in filling pressure will reduce lymphatic flow; thus edema occurs (Laine et al., 1986). This explains the observation made by Kowalski et al. (1989) that patients with pulmonary capillary wedge pressure (PCWP) greater than 14 mmHg during CPB showed significant increase in EVLW.

Other studies have shown that the type of fluids has no influence in EVLW formation. Ley, Miller, Skov, and Preisig (1990), in an experimental design of fresh post-CABG patients compared the efficiency of fluid management between crystalloid and colloid. They found that the crystalloid group required a larger fluid volume to achieve hemodynamic stability with lower filling pressures. On the other hand, the colloid group required a smaller fluid volume to achieve better hemodynamic parameters with higher filling pressures. The counterbalancing effect of each fluid explained that the type of fluids does not contribute to EVLW formation. Another variation of this study was done by Marelli et al. (1989). In an experimental design, they determined the benefit of adding 50 gm Albumin to the prime solution during CPB. They found no significant difference in clinically monitored cardiorespiratory and renal parameters between the two groups, identical findings in fluid volume and filling pressures to those of Ley et al. (1990). No clinical evidence of pulmonary edema was observed in any of the studied patients, except that in the Albumin group the $P(A\text{-}a)DO_2$ was higher than in the other group. This is further supported in animal studies where elevation in EVLW was not reproducible by lowering the plasma COP (Kowalski et al., 1989). Instead, when the reduced COP was combined with an elevated filling pressure, an increase in EVLW became evident.

In an attempt to determine that an increase in EVLW is not related to an increase in capillary permeability from aortic cross-clamping, Kowalski et al. (1989) measured EVLW with a special catheter during aortic cross-clamping in nine abdominal aortic surgery patients. Normal filling pressures were maintained during the surgery, and the authors found no significant increase in EVLW in any of the studied patients. The strong implication is that the determinant for increase in EVLW is elevated filling pressures.

In summary, CPB has been used widely since its inception in the 1950's, but the mechanisms that causes the pulmonary dysfunction in the post-operative period is still obscure. A general belief is that activation of the complement system triggers a cascade of events with the final outcome of multiple organ dysfunctions, the respiratory system appearing to be most vulnerable.

Effect of the CABG Surgical Procedure on Pulmonary Function

The two major determinants of atelectasis in post-operative CABG patients have been alluded to in the above discussion. The third important determinant of atelectasis in post-operative CABG patients is the actual surgery, which may interfere with the respiratory muscle function. It is indisputable that incidence of atelectasis is high in this group of patients. Chest radiography is commonly used for identifying post-operative atelectasis, with most apparent changes occurring within 24-48 hours (Chin et al., 1986). In some patients, atelectasis is attributable to diaphragmatic paralysis (Good et al., 1979). There is considerable variability in the reporting of the incidence and severity of diaphragmatic elevation or paralysis after cardiac surgery because of diagnostic difficulties (Bogers, Nierop, Bakker, & Huysmans, 1989). The paralysis is linked to the phrenic nerve damage during the surgery, and most of the reports focus on topical cardiac cooling as a major contributor to post-operative phrenic nerve paralysis (Good et al., 1979; Curtis et al., 1989). The new method of cardiac cooling with ice slush in the pericardium for myocardial preservation has been shown both clinically and experimentally, to be associated with dysfunction and damage of the phrenic nerve and sequential left lower lobe atelectasis (Robicsek et al., 1990; Curtis et al., 1989).

Robicsek et al. (1990) used a canine model to find out the degree of cold exposure sufficient to inflict hypothermic phrenic nerve injury and sequential diaphragmatic paralysis. The experiment showed that phrenic nerve conduction ceased at 3°C and did not return within four hours; two types of nerve preparation were found resistant and sensitive to cold injury. When nerves were exposed to 4°C, 35% of the signal recovered within four hours. Application of temperature below this level creates a situation favourable to the development of phrenic nerve paralysis. The vulnerability of the phrenic nerve to cold injury is because of its anatomic location. It lies within the layers of the fibrous pericardium, and motor fibre is most susceptible to cold injury. The higher sensitivity of the left versus the right phrenic nerve is due to the anatomy of the heart, where most of the newly applied and melting slush accumulates on the left side. Robicsek et al. speculated that the clinical outcome of the frostbitten phrenic nerve syndrome is variable among patients; this may be that some patients have resistance nerves while others have cold-sensitive nerves.

Curtis et al. (1989) report that an elevated hemidiaphragm was high in patients who had topical ice slush cooling; the incidence increased when a mammary graft was used as well. They conclude that the probability and duration of the damage will depend on the depth and time limit of the topical cardiac hypothermia and the individual anatomy of the patient. The application of protective devices will decrease but will not eliminate, the danger of cold injury to the phrenic nerve.

Reviewing chest X-rays, Wilcox et al. (1988) found evidence of left lower lobe atelectasis in 50 out of 57 patients and right lower lobe atelectasis in 35 out of 57 patients in the post-operative period. Atelectasis was more severe for the left lung than for the right in all post-operative days. In this series, five of the 52 patients developed an unequivocal abnormality in the left phrenic function; only three out of the five had hemidiaphragmatic paralysis. In 27 out of the 47 remaining patients, there was a decrement in the left phrenic nerve action potential to stimulation post-operatively. The electrophysiology study of the phrenic nerve function on post-operative day one might exclude patients with transient phrenic nerve injury.

An internal mammary artery graft (IMA) as a conduit has demonstrated a longer patency rate; thus its popularity continues despite known compromises to pulmonary function. Jansen and McFadden (1986) document the fact that IMA graft is linked to an increase in intraplumonary shunting, a decrease in PaO_2, and an increase in atelectasis. The exact mechanism of why an IMA graft causes more pulmonary dysfunction is unclear, but there is speculation that the harvesting of the left IMA graft is more difficult, the incidence of pleurotomy is high, and the placement of a pleural drain is necessary (Jenkins, Soutar, Forsyth, Keates, & Moxham, 1989). In a comparison of post-operative lung functions between patients with saphenous vein grafts (SVG) to those who had both SVG and LIMA grafts, Jenkins et al., (1989) found that lung volume decreased in both groups with maximal decline on day two. It remained low even on day four. Greater reduction in lung volume, more analgesic requirements (Mailis et al., 1989), and a higher frequency of chest infection were observed in the group with an IMA graft.

In another study, Berrizbeitia et al., (1989) examined lung function in 55 CABG patients six to eight weeks after their surgery. They found that a median sternotomy incision along with an IMA graft is associated with greater impairment of pulmonary mechanics than SVG with the same incision. This is related to the reduction in chest wall compliance and the blood supply to intercostal muscles. All the studies so far show that atelectasis may be caused by several intra-operative factors. However, topical ice slush and IMA graft are considered to be important contributors to atelectasis.

In summary, pulmonary dysfunction observed in the post-operative period may be due to the summation effect of the anaesthetic agent, the CPB, the surgical procedure, and the age of the patient. The dysfunction may be manifested in increase intrapulmonary shunting, $\dot{V}/\dot{Q}$ inequality, and atelectasis which results in hypoxemia.

Positioning and Lung Diseases

Alteration in the $\dot{V}/\dot{Q}$ ratio is the most common cause of hypoxemia. The effect of positioning has the potential of alternating this ratio and can be used as a therapeutic measure to minimize hypoxemia.

Oxygenation may improve in patients with unilateral lung disease when the good lung is placed in the dependent position (Robichaud, 1990). Dependent positioning of the good lung enhances perfusion to better ventilated areas, thus matching the $\dot{V}/\dot{Q}$ ratio. Some authors suggest that for patients with unilateral lung disease, positioning should be adjusted according to the underlying lung pathology to achieve optimal arterial oxygenation (Hall & Wood, 1987; Robichaud, 1990). However, the effect of positioning on oxygenation in bilateral lung disease or diffuse lung disease is still obscure (Robichaud, 1990). In fact, when patients with bibasilar lung disease are placed in an upright position, the result may be significant hypoxemia. This illustrates that positioning is a consideration for optimal gas exchange.

In a review of studies concerning immobility and pulmonary complications, Tyler (1984) reports that the long-term effect of strict bedrest in young, healthy men resulted in a decline in PaO_2 and a widening $P(A-a)DO_2$. This observation was further investigated in animal studies by Ray et al. (1974). They compared the effect of turning on overhydrated dogs. Nine dogs were divided into three experimental groups: the control group was kept in one lateral position for the duration of the study, the second group was turned every half-hour to alternate lateral positions, and the third group was turned hourly. All the dogs were anaesthetized, ventilated, and systematically overhydrated to 25% of extracelluar fluid excess (ECF). The reason for the deliberate fluid overload was to test if this was an important co-factor in shunting. However, the authors failed to report the findings on all the dogs; instead, they discussed one dog in each group. This limited the validity of the results. Despite this shortcoming, the findings described by the authors are dramatic. In the immobile dog, the PaO_2 rapidly deteriorated and a 40% shunting was noted halfway through the study. At postmortem, the dependent lung weighed twice that of the non-dependent lung, with subpleural haemorrhages and atelectasis. In the hourly mobilized dog, the PaO_2 of each lung followed a ziz-zag pattern indicating desaturation when in the dependent lung and oxygenation in the non-dependent lung. The overall PaO_2 was relatively constant in this group, with shunting below 30% throughout the study. The postmortem study revealed the right lung weighed 50 gm less than the left; this information is meaningless since the authors did not report the position the dog was in prior to death.

Both lungs had scattered areas of atelectasis and subpleural haemorrhage. In the half-hourly turned dog, there was a progressive improvement in PaO_2 and a decrease in shunting to physiological level despite vigorous overhydration. At termination, the right lung was 40 gm less than the left with scattered pathological changes and frothy sputum in the trachea. Engorgement of lymph nodes was observed in all the dogs. This study shows that in the presence of overhydration, frequent redistribution of blood flow and ventilation appear to preserve pulmonary function. The authors conclude that with fluid overload, interstitial edema occurs in dependent regions of the lung and blood flow is high in these areas, resulting in shunt and hypoxemia. In the same paper, Ray et al. (1974) describe two ventilated patients in which frequent turning was implemented. The first patient had respiratory failure; following the turning regimen, shunting in this patient decreased from 34% to 12% and PaO_2 climbed from 50 mmHg to 93 mmHg. In the second patient, the pulmonary vein was cannulated with a small catheter during hiatus hernia repair via the left thoracotomy approach for the purpose of blood sampling. Sampling was done in arterial, pulmonary, and central venous blood for gas analyses. Data on these gases were not presented, but the authors conclude that better pulmonary venous oxygen was obtained when the operative side was up than when it was down. Does frequent turning interfere with gravitationally induced interstitial edema, does it causes equalization of $\dot{V}/\dot{Q}$, or does it enhance alveolar stability?

The protective function of frequent turning is further verified in an observational study of spinal cord injury patients who were placed on a kinetic treatment table (KTT) (a bed which rotates at an arc of 124° continuously). The study reported a 1.9% respiratory mortality rate (Tyler, 1984). Pulmonary mortality in this group of patients varied from 27% to 100% (Tyler, 1984), and the striking reduction may be attributable to turning. It would lend more power to the study if there had been a control group of spinal cord injury patients in conventional beds. This deficiency was recently rectified in two similar studies done by Demarest, Schmidt-Nowara, Vance, and Altman (1989) and Gentilello et al. (1988). Demarest et al. compared the KTT to conventional beds in mechanically ventilated trauma patients to determine the incidence of atelectasis and pneumonia. The only difference between the groups was the frequency of turning: the conventional bed patients were turned every two hours and the KTT patients were rotated continuously except for care. The duration of the study was one week. No significant changes were found in any parameters tested between the groups. However, a subset analysis of the data showed that KTT can significantly prevent pulmonary complications in bedridden patients. This was only applicable to patients who had an initial normal chest X-ray. However, in those patients with abnormal chest X-rays at the start of the study, the KTT demonstrated no protective benefit in minimizing respiratory complications. The major limitations of this study were the time allowance to determine the development of and recovery from pulmonary complications and the single criterion in establishing pulmonary complications.

Gentilello et al. (1988) replicated the study by Demarest et al. (1989), but this group of researchers found significant differences between the two groups, with a higher incidence of atelectasis in the conventional bed group than in the KTT experimental group. Upon closer examination of the data, the percentage of normal X-rays was higher in the experimental group, with an uneven distribution of smokers among the groups. The subjects in the experimental group were rotated slightly more than 50% of the time. Is increasing the frequency in intermittent turning as effective? Gentillelo et al. supply some insight into the reason for improvement in gas exchange with frequent turning. They explain that gravity can supplement the effectiveness of the mucociliary apparatus in removing pulmonary secretion and in preventing stasis of secretion to dependent regions of the lung. Other researchers have looked at alternatives to improve pulmonary functions other than the frequency of turning. Studies have been done to maximize $\dot{V}/\dot{Q}$ matching to enhance PaO_2 by manipulating different body positions (Norton & Conforti, 1985). Piehl and Brown (1976) studied five ventilated patients with ARDS in circo-electrical bed to learn the effect of supine and prone positioning. They found significant improvement in PaO_2 values in the prone position. However, deterioration in PaO_2 was found when these patients were left in the position for more than four hours; improvement was noted again after repositioning. Douglas, Rehder, Beynen, Sessler, and Marsh (1977) replicated Piehl and Brown's study in a conventional bed; the findings of the study were similar. Regular turning between the supine and the prone positions demonstrated a higher PaO_2 in the prone position with no significant variation in ventilation. The observed changes might have been due to $\dot{V}/\dot{Q}$ and FRC modification. In 11 artificially induced ARDS canines, Albert, Leasa, Sanderson, Robertson, and Hilastala (1987) examined the physiological mechanism that causes improvement in oxygenation in the prone position. In the prone position, PaO_2 climbed with a simultaneous fall in shunting, and these improvements could not be attributable to changes in FRC, cardiac output (CO), regional diaphragm motion, or pulmonary vascular pressure. These researchers imply that reduction in ventilation occurs only when the lower lobes are dependent. However, these animals were all ventilated with a tidal volume of 20 ml/kg and 100% FiO_2; this was not considered in the analysis of the results. The lack of a control group in this study makes it difficult to establish the impact of confounding variables on the criterion.

In a review of studies with positioning and oxygenation, Grosmaire (1983) reports that the possible mechanisms for improvement in oxygenation in the prone position may be an increased FRC, redistribution of edema fluid and enhanced lymphatic drainage, redistribution of pulmonary blood flow, and improved bronchial secretion clearance. These possible explanations were challenged in a recent study which focused on the prone position and ARDS patients. Langer, Mascheroni, Marcolin, and Gattinoni (1988) found a high prevalence of dense areas in bilateral posterior-basal regions in ARDS patients. The study was to determine the mechanism of the prone position to improve oxygenation. Thirteen mechanically ventilated, sedated, and paralysed ARDS patients were studied. The parameters being evaluated were hemodynamics, blood gases, and the thoracic CT scan. They classified patients into two groups: responder and non-responder. The criterion to be a responder was an increase in 10 mmHg PaO_2 from baseline after repositioning. Each patient was turned every two hours between supine and prone positions, with arterial blood gas analyses at designated time intervals. Data showed an overall increase in PaO_2 in the prone position with no significant changes in $PaCO_2$ and hemodynamic parameters. Eight patients were responders and five were non-responders to the positioning. In fact the non-responders showed deterioration in the prone position. The shunt fraction was not statistically different between the two positions. In the CT scan study, the prone position caused almost a total clearing of the posterior-basal density seen in the supine position, but new densities appeared over time in previously clear anterior areas. In a subsequent part of the study, the researchers studied the responders over time in these two positions and noticed that general improvement in PaO_2 and a decline in shunting persisted even when the supine position was resumed. The possible mechanisms involved in improvement of oxygenation are many. Blood flow redistribution is considered as an important factor in matching $\dot{V}/\dot{Q}$, but the CT scan also demonstrated redistribution of the dense areas to the apical area over time. An increase in FRC in the prone position is another possible mechanism, but this does not explain the persistent improvement in oxygenation over time when the supine position was resumed. The alteration in hydrostatic pressure so that edema fluids collect in dependent areas may explain the initial rise in oxygenation after the prone position, but this should reverse over time to worsening oxygenation. The explanation of gas redistribution in mechanical ventilation and in positioning results in re-expansion of previously collapsed airways in non-dependent lung regions, does not give reasons for continual improvement in oxygenation associated with the prone position. In the presence of a non-responder group, the exact mechanisms responsible for improvement in gas exchange are still obscure.

The prone position may not be feasible for all patients, especially for critically ill patients. Lateral positioning is popular in nursing practice, but positioning patients without regard to underlying pulmonary pathology may in fact compromise arterial oxygenation. Zack et al. (1974) compared the effects of lateral positioning in healthy volunteers and patients with lung diseases. All subjects were breathing spontaneously. Data showed that in patients with unilateral lung disease, PaO_2 was significantly higher when the healthy lung was dependent than when the contralateral lung dependent. In patients with bilateral chest X-ray abnormality, PaO_2 was generally 9 mmHg higher in the right lateral than the left lateral positions, but the difference was non-significant. On the other hand, in healthy subjects and in patients with normal chest X-rays, PaO2 did not change significantly in either lateral position. $PaCO_2$ was not affected by the positions. The proposed mechanism for the observed changes in oxygenation then is modification in the $\dot{V}/\dot{Q}$ relationship. The findings of a higher PaO_2 when lying on the right side in presence of bilateral lung disease suggests that impairment of gas exchange is uneven and there was a greater compromise in gas exchange with lying on the left side. Zack et al. (1974) speculate that the difference might be anatomical. The left lung is subjected to more compressive forces from the heart and the mediastinal structures. These structures may reduce the left lung volume when the left lung is dependent than when the right lung is dependent.

Remolina et al. (1981) replicated Zack et al.'s (1974) study in nine patients with unilateral lung disease. One patient was mechanically ventilated and eight others were on supplementary oxygen. They compared the PaO_2 value in the three positions: supine, right and left lateral. The findings were in keeping with those of Zack et al. (1974). A graded difference was observed in the PaO_2 value, with the highest PaO_2 value when the healthy lung was in the dependent position, then the supine position and the lowest PaO_2 value when the diseased lung was dependent. Seven of these patients were followed and the study was repeated after resolution of the lung disease. The researchers found that positional hypoxemia was resolved. Again the mechanism was believed to be $\dot{V}/\dot{Q}$ inequality. The authors explained that when the diseased lung is dependent, gravitational effect causes preferential blood flow to the dependent region while ventilation can not increase appropriately because of the underlying pathology. The inability to increase ventilation in the diseased lung may be due to the filling of the alveoli with exudate and the reduction in lung volume, which leads to reduction of volume below FRC and increases the risk of airway closure in the dependent regions. Similar findings of improved oxygenation with the "good-lung dependent" but with different pulmonary abnormalities have been reported (Dhainaut et al., 1980; Seaton, Lapp, & Morgan, 1979).

Sonnenblick et al., (1983) studied eight patients with unilateral pleural effusion, and the good-lung dependent theory still persisted. However, in patients with larger pleural effusion, the positional hypoxemia was less than in those with smaller pleural effusion. The authors explained that large pleural effusion decreases lung volume and renders the intrapleural pressure less negative. This may lead to airway closure on the effusion side and decreased ventilation. Reduction in lung volume causes the compression of extra-alveolar vessels, thus increasing the pulmonary vascular resistance (PVR) of the affected side and decreasing perfusion despite the gravitational effect of perfusion. Furthermore, alveoli hypoxic vasoconstrictor response may also contribute to decreased blood flow to the dependent lung with pleural effusion. The combined mechanical and local reflex produces a shifting of blood to the non-dependent lung. This hypothesis was confirmed by a perfusion lung scan in the patient with the largest effusion in the study. A gross reduction in total perfusion was evidenced in the pleural effusion lung.

Gillespie and Rehder (1989), in a case study of a patient with moderate unilateral pleural effusion who was also ventilated with PEEP, found insignificant changes in oxygenation, the $\dot{V}/\dot{Q}$ relationship, and $PaCO_2$ in both lateral positions. Anthonisen and Martin (1977) provide the possible explanation by having used radioactive Xenon gas in a patient with unilateral pleural effusion. They found that the total lung capacity (TLC) decreased in the effusion side but the relative regional volume was unchanged as compared to the contralateral side. Their proposed hypothesis is that with pleural effusion there may be displacement of the diaphragm. Conflicting results from Gillespie and Rehder's case report (1989) and Sonnenblick et al.'s (1983) may be attributed to the mode of ventilation.

All of the above studies imply that in a spontaneously breathing patient, better oxygenation can be achieved by positioning the "good lung" dependent to augment the $\dot{V}/\dot{Q}$ ratio. However, in mechanically ventilated patients with positive end expiratory pressure (PEEP), positional hypoxemia may not exist, as implied by Gillespie & Rehder (1989).

Positioning and Mechanical Ventilation

Mechanical ventilation (MV) with PEEP is used in patients with respiratory failure because of its ability to recruit underventilated alveoli by increasing FRC (Glauser, Polatty, & Sessler, 1988). MV results in increased lung compliance, a reduced low $\dot{V}/\dot{Q}$ region, and improved PaO_2 (Hubmayr, Abel, & Rehder, 1990). Prophylactic PEEP does not alter the course of the underlying disease. On the contrary, PEEP can increase the water content of the lung (Hubmayr et al.,1990).

Basically, mechanical ventilators function as substitutes for the bellow action of the diaphragm and the chest cage. In order to move air into the lung, pressure must be high enough to overcome the airway resistance and the elastic properties of the lungs and the chest wall. During positive pressure ventilation (PPV), respiration becomes passive and gas distribution is altered by this mode of ventilation. In PPV, gas distribution decreases to the dependent region of the lung (Nunn, 1987; Robichaud, 1990; Krayer et al., 1989; Baehrendtz & Klingstedt, 1984). This is primarily because of the hydrostatic forces opposing the displacement of the diaphragm. In the lateral position, the dependent diaphragm rises high into the thorax because of the displacement of the abdominal content. This same pressure will oppose the descent of the diaphragm during respiration (Nunn, 1987). Consequently, the non-dependent areas ventilate better than the dependent areas.

The current belief is that a high tidal volume (12 to 15 ml/kg) and a slow rate with PEEP will prevent airway closure and enhance lung compliance, thus improving gas exchange. But high tidal volume and PEEP are associated with high peak airway pressure, and this has been demonstrated to be detrimental on the lung parenchyma (Haake, Schlichtig, Ulstad, & Henschen, 1987). Healthy animals when subject to a peak airway pressures at the 35 cm H_2O ranges for more than two hours showed such adverse effects as bilateral pulmonary atelectasis, alveolar edema, hyaline membrane formation, and interstitial haemorrhage (Lee, Helsmoortel, Cohn, & Fink, 1990). Kolobow et al., (1987) in a study of post-anaesthetic healthy sheep ventilated at two different tidal volumes and peak airway pressure, investigated the effect of this mode of ventilation. The control group was ventilated with 8-15 ml/kg, 6-8 cm PEEP and peak airway pressure of less than 20 cm H_2O. The experimental group was ventilated with a high tidal volume to achieve a peak airway pressure of 50 cm H_2O. There was no difference in the initial course between the groups. In the experimental group, after a few hours of ventilation, there was a progressive deterioration in PaO_2, FRC, and static lung compliance with a high mortality rate. An autopsy showed gross alteration in the lung structure. This study illustrates the potential danger associated with high tidal volume and high peak airway pressure. In an earlier study, Greenfield, Ebert, and Benson (1964) found that short-term overinflation of the lung with a tidal volume of 14 ml/kg results in a delay in the appearance of abnormalities in the surfactant system. At 24 hours post-hyperinflation, there was an increase in surface tension and surface tension returned to baseline at 48 hours. It appears that type II cells in the alveolar lining are vulnerable to mechanical trauma.

Some authors state that low tidal volume and higher frequency ventilation will result in lower airway pressure, thereby minimizing potential barotrauma of the lung (Lee et al., 1990). They compared low (6 ml/kg) to high (12 ml/kg) volume ventilation with PEEP in 101 trauma and aortic abdominal surgery patients. The data revealed that low tidal volume resulted in significantly lower peak airway pressures and PaO_2/FiO_2 ratio. The difference in the PaO_2/FiO_2 ratio was 34, which was not clinically significant. However, the incidence of pulmonary infection was higher in the large tidal volume group than in the low, with percentages of 17.9% to 4.3% respectively. This demonstrates that lower tidal volume with PEEP may be as effective as higher tidal volume with or without PEEP. The reason for the lower incidence of infection in the low volume group is unclear; it may be related to fewer microscopic barotrauma (Lee et al., 1990). The limitation of this study is the lack of X-ray comparison between the two groups, as the animal studies suggest that high tidal volume with high peak airway pressure may cause atelectasis and parenchyma changes. Utilizing large tidal volume and PEEP to re-expand collapsed lung regions only causes overexpansion of the uninvolved lung and produces barotrauma to the normal region, increase the extravascular fluid accumulation, reduce lymphatic flow, and displace perfusion to regions of less vascular resistance (Bevan, 1990; Kahn, Koslow, & Weinhouse, 1988). The net effect is an increase in both dead space ratio and pulmonary venous admixture.

In another similar study of 10 obese gastric bypass patients, Eriksen, Andersen, Rasmussen, and Sorensen (1978) compared cardiorespiratory functions of high tidal volume (35% above normal) and 750 ml tidal volume with PEEP. The mean weight of these patients was 129 kg. The findings revealed that both methods were equally effective in enhancing PaO_2 and decreasing $P(A-a)DO_2$, consistent with an earlier study by Visick, Fairley, and Hickey (1973). The normal tidal volume with PEEP group showed higher lung compliance but the cardiac index was significantly lowered. Positive pressure ventilation with PEEP did influence cardiac function. The increase in intrathoracic pressure during positive pressure ventilation causes a decrease in venous return as a result of compression in the large blood vessels and consequently decreases CO. Therefore, in patients who are hypovolemic, this decrease in CO is subsequently reflected in decreased arterial blood pressure (BP).

Szold, Pizov, Segal, and Perel (1989) investigated the relationship between tidal volume and hydration status in 10 dogs. Three conditions were sequentially induced in each of the dogs in the study: normovolemic, hypovolemic (30% of estimated total blood volume), and hypervolemic. A comparison of tidal volumes was made between 15 ml/kg to 25 ml/kg in each of these volemic conditions. In the hypervolemic group there was a minimal variation in hemodynamic parameters between the two tidal volumes. A significant variation in hemodynamic parameters was found in the hypovolemic group with both tidal volumes. It is unfortunate that no indicators of oxygenation were obtained during the study. The authors suggest that when high tidal volume and PEEP are utilized in PPV, the hypervolemic state may inhibit the fluctuation in perfusion seen in the hypovolemic group, thus minimizing the $\dot{V}/\dot{Q}$ changes and subsequent oxygenation.

Use of PEEP in conjunction with high tidal volumes may worsen oxygenation by redistribution of blood flow to non-ventilated regions, thus increasing the venous admixture (Kanarek & Shannon, 1975; Johnston, Vinten-Johansen, Santamore, Case, & Little, 1989). That mechanical ventilation with PEEP influences the $\dot{V}/\dot{Q}$ ratios has been repeatedly reported by many researchers (Cheney, 1972; Colgan & Marocco, 1972; Thornton, Ponhold, Bulter, Morgan, & Cheney, 1975; Greenfield et al., 1964). Does positioning change the $\dot{V}/\dot{Q}$ ratio and oxygenation? Norton & Conforti (1985) cite a case report in which a patient suffered injury to the left lung and required ventilatory support. This patient was mechanically ventilated with 15 ml/kg with alternating no PEEP and 5 cm H_2O of PEEP. Data collection occurred over three days and the patient was randomly placed in one of the three possible positions, with clinical parameters and blood gases taken in each position after 30 minutes with PEEP and no PEEP. During no PEEP ventilation, positional hypoxemia existed with the injured lung-dependent and increased shunting with no significant changes in CO or PVR. When PEEP was added, there was an improvement in oxygenation in all three positions, but a 15% drop in PaO_2 and a deterioration in shunting was observed when the "bad lung" was dependent as compared to the other two positions.

Ng and McCormick (1982) reviewed a study in which five normal subjects' $\dot{V}/\dot{Q}$ distribution was evaluated during spontaneous and intermittent positive pressure ventilation (IPPB) using radioactive Xenon gas. The study demonstrated that the ventilation pattern between spontaneous and IPPB was opposite and that the blood perfusion was unchanged. During IPPB, the dependent lung regions are under-ventilated whereas the non-dependent lung regions are over-ventilated. This increases the non-homogeneity of ventilation within the lung. In the supine position, ventilation and perfusion are lower to bibasilar lung regions than in the upper regions, providing evidence that artificial ventilation and positioning contribute to $\dot{V}/\dot{Q}$ maldistribution in the healthy individual.

How does PPV, PEEP, and positioning affect $\dot{V}/\dot{Q}$ distribution in the presence of lung pathology? Gillespie & Rehder (1987) investigated this question in four long-term ventilated patients with unilateral lung disease, using gas chromatography to measure $\dot{V}/\dot{Q}$ distribution in different positions: supine, right, and left laterals. When the "good lung" was in the dependent position, oxygenation improved significantly without associated changes in $PaCO_2$. The improvement in oxygenation was mainly due to a decrease in right to left shunting by reducing the low $\dot{V}/\dot{Q}$ regions and better matching of $\dot{V}/\dot{Q}$. Gas distribution favours the non-dependent regions. In contrast, blood flow to the lung is mainly determined by gravitational forces, but flow can also be influenced by non-gravity related factors such as CO, alveolar pressure, and alveolar hypoxemic vasoconstrictor response (HPV). However, gravitational forces overcome any of the above factors. A number of small sample-size studies of PPV patients with unilateral lung disease duplicated the findings that the "good lung" dependent improves gas exchange (Rivara, Artucio, Arcos & Hiriart, 1984; Ibanez et al., 1981; Hasan, Beller, Sobonya, Heller, & Brown, 1982).

Nelson and Anderson (1989), in a repeated measures design, studied cardiorespiratory function in 10 patients with symmetrical lung disease in four different positions: supine, right, left, and continuous rotation of these three positions. Hemodynamics and respiratory parameters were collected after a 30-minute stabilization period in each position. All patients were ventilated with a constant FiO_2 and PEEP. The findings showed variable response to positioning. Four patients showed a significant decline in PaO_2 in both lateral positions when compared to supine; continuous rotation of these four patients restored the PaO_2 to the baseline value.

Chest x-rays indicated no asymmetric disease. The remaining six patients, the mean PaO_2 increased on lateral positions as compared to supine. Of these six patients, five had a higher mean difference between the right and left lateral positions. Subsequent X-ray examination of these five patients indicated asymmetrical lung infiltrate. Patients who responded by improving in PaO_2 in one position did not benefit from continuous rotations as indicated by no changes in the PaO_2 value from the baseline. This study shows that in symmetrical disease, positioning to optimize oxygenation needs to be individualized. Furthermore, it appears that if lung disease is symmetrical, then the supine position may result in better gas exchange.

This and other studies discussed above show that positioning has a role in improving PaO_2; however, the small sample in all of these studies makes it difficult to isolate the contributors to the improvement in gas exchange associated with posture. In general, the "good lung-dependent" manoeuvre to enhance PaO_2 and to reduce shunting appears repeatedly in patients who are breathing spontaneously or are mechanically ventilated. In addition, there is supporting evidence that ventilation with PEEP is superior to ventilation without PEEP, but large tidal volumes with PEEP is not any different than normal tidal volumes with PEEP. Alteration in CO with PEEP may potentially alter the $\dot{V}/\dot{Q}$ ratio, therefore the patient's hydration status is important during PPV with PEEP.

Positioning and Post-operative CABG Surgery

Interest in positioning in post-operative CABG patients stems from the study by Chulay, Brown, and Summers (1982) in which patients who had CABG were not turned for 18-24 hours in their own institution. Upon further investigation, the authors found that the practice of immobilization of CABG patients in the supine position for 24 hours was a norm in 50% of the university hospitals they surveyed. The reasons for not turning these patients were the fear of increasing oxygen consumption and desaturation. Chulay et al. in a two-month retrospective chart review, found that 84% of patients developed atelectasis within the first 72 hours post-CABG. The common post-operative complications for this surgery are pyrexia, atelectasis, and hypoxemia.

In order to determine the possible benefits of early mobility in minimizing these complications, Chulay et al. (1982) in an experimental design, studied 35 ventilated post-CABG patients. These patients were randomly assigned into two groups: the immobile group and the mobile group, which was turned systematically every two hours immediately after surgery. Hemodynamic parameters, temperature, and blood gases were monitored. $P(A-a)DO_2$ was measured at an FiO_2 of 1.0 on admission to the unit and at discharge. Chest x-rays followed for three days after surgery. No significant differences were found in all the parameters recorded except temperature, intubation time, and ICU stay, which were significantly lower in the mobile group. There were no differences in the chest x-ray abnormalities between groups, with 70% incidence of atelectasis. $P(A-a)DO_2$ on admission and discharge was not significantly different, but the immobile group had a mean difference of 167 mmHg and the mobile group had a mean difference of 147 mmHg. This might indicate that the severity of atelectasis was worse in the immobile group. PaO_2 was drawn every four hours; no difference was found between groups. The lack of change in this parameter may be due to the fact that blood gases were done in a supine position in both groups, or there may have been errors in comparing PaO_2 when patients were on variable oxygen concentrations. Systematic turning in this study clearly showed benefits in the reduction of post-operative fever, shorter intubation time and ICU stay. Changes in PaO_2 might have been demonstrated by Chulay et al.' study if blood gases had been drawn at each position in the mobile group. This may prove a further advantage in early mobilization and the importance of the position the patient assumes in the presence of atelectasis.

Banasik et al., (1987) advocate that immobility in CABG patients may compound the inherited pulmonary dysfunction associated with the surgery. The detrimental effects of immobility can easily be minimized by turning. In an attempt to demonstrate the benefits of early turning in respiratory function, Banasik et al. in a repeated measures design, investigated 60 post-operative CABG patients and the effect of three positions: supine, 45° left, and 45° right lateral on oxygenation. Arterial blood gases were drawn at each position 10 minutes after repositioning. All patients were ventilated at the time of the study. The data showed no significant differences in the turning sequencing, the repeated turning, the respiratory rate, or the $PaCO_2$. In regrouping the data into specific positions, significant differences were noted in PaO_2. Supine and right lateral positions had a higher mean PaO_2 value than the left lateral position. The change in SaO_2 level was non-significant in all positions, even in positions where the PaO_2 level was low. This is because of the flat portion in the oxyhemoglobin curve where a fluctuation of PaO_2 will not significantly change the SaO_2. Seventeen percent of the patients had LLL atelectasis, 23% had bilateral atelectasis, and no patients had RLL atelectasis. When patients with LLL atelectasis were removed from the analysis, there was no difference between right and left lateral positions. In the presence of bilateral atelectasis, positioning had no influences on oxygenation. This study confirms the findings of research done with patients having unilateral and bilateral lung diseases (Zack et al., 1974). It has been demonstrated that frequent turning may prevent formation of a low $\dot{V}/\dot{Q}$ region in the lung (Ray et al., 1974), a confounding variable in which Banasik et al. have not considered in their study.

Gillespie, Didier, and Rehder (1990), in an attempt to differentiate the major mechanisms responsible for hypoxemia in post-operative cardiac surgery patients, used the multiple inert gas technique to study $\dot{V}/\dot{Q}$ distribution and shunt following aortic value replacement in eight ventilated patients . Patients in the study all had normal pulmonary functions pre-operatively. All patients were studied in the supine position and three were also studied on the right side. The data showed $\dot{V}/\dot{Q}$ abnormalities in all patients when compared to pre-operative data. All patients had a right to left shunt which varied from 2.4% to 14.4% and significant areas of low

$\dot{V}/\dot{Q}$ ratio. In those patients who were placed in a right lateral position, the expired minute ventilation and left atrial pressure were higher than their supine values; improvement in PaO_2 was without concurrent changes in $PaCO_2$. This improvement in oxygenation could not be attributed to a decrease in shunting or a decrease in low V/Q ratio, because two of the three patients had an increase in shunting and low $\dot{V}/\dot{Q}$ ratio. Instead, the data showed more uniformity in overall ventilation and perfusion matching. The authors indicate that this study was conducted when dysfunction in gas exchange was maximal. This is based on previous studies in which pulmonary abnormalities were detected by x-ray on day one after heart surgery, and gas exchange abnormalities were maximal at 24 hours and persisted for 48 to 72 hours. This study shows that positional improvement in oxygenation appears not to be associated with improvement in shunting or low $\dot{V}/\dot{Q}$ ratio.

Shively (1988), in a repeated measures experimental design, studied the effect of positioning, frequency of positioning, and timing of measurements of $S\bar{v}O_2$ in 30 extubated post-CABG patients. These patients were randomly assigned to either the hourly or the two hourly turn group, and all of them were turned at the same sequence: right lateral HOB 20°, supine HOB 45°, left lateral HOB 20°, and supine HOB 20°. Baseline parameters for comparison were obtained when the patients were in the last supine position. No significant differences in SvO_2 were found in the different positions and frequency of turning. There were significant differences in SvO_2 between 0 min-15 mins, 0 min-1 hr, 0 min-2 hrs, and no significant differences noted in other paired comparisons. This shows that a transit drop in SvO_2 occurred with turning, and this drop was observed by the author to last only five minutes. The rapid desaturation of mixed venous oxygen could be due to an increase in peripheral oxygen extraction secondary to increased activity. The author concludes that positioning does not change oxygenation. A major limitation of this study is the use of SvO_2 as an indicator of oxygenation. SvO_2 is dependent on the interaction between oxygen consumption, cardiac output, SaO_2, and haemoglobin concentration. In re-arranging the Fick equation the following is obtained:

$$SvO_2 = SaO_2 - [\dot{V}O_2/(Hgb \times 1.34 \times \dot{Q})]$$

The dependency on the above components makes SvO_2 a non-specific parameter to be used as a single parameter in determining oxygenation. SvO_2 reflects the mean oxygen utilization from various organs and is not a reflector of oxygenation at the lung level (Jamieson et al., 1982). In other words, SvO_2 reflects the supply and demand relationship of oxygen (Ahrens, 1990). The erroneous use of SvO_2 as a dependent variable in Shively's study limits the usefulness of the results.

Noll and Fountain (1990), in a repeated measures experimental design, examined the effect of different bedrest positions on patient oxygenation. SvO_2 again was utilized as an indicator of oxygenation in 30 ventilated post-CABG patients. These patients were randomly assigned to three groups: 20° headrest elevation, 40° headrest elevation, and supine with no headrest elevation. Baseline hemodynamic and SvO_2 data were collected in the supine flat position prior to entry into each group. In the treatment position, data was collected at 0, 5, 15, and 30 minute times; then the flat supine position was assumed and data collection was repeated. The data showed no significant difference in treatment effect, time, or the interaction of these two factors. The premise of this study is that SvO_2 may change with changes in headrest elevation because of alteration in cardiac output, $\dot{V}/\dot{Q}$ ratio, and oxygen consumption. Obviously it is not oxygenation the authors are interested in but the balance of oxygen supply and demand with different headrest elevations. The non-significant findings may be due to the activation of the compensatory mechanisms. There is a question of validity in comparing SvO_2 when patients were receiving supplemental oxygen at different concentrations.

Banaski (1990) reports in an abstract that there are no significant differences in positioning and oxygenation in post-operative CABG patients. Forty ventilated post-CABG patients were studied in three positions: supine, right, and left lateral positions. A simultaneous arterial and mixed venous gas were drawn in each of the three positions. There was no significant difference in mixed venous partial pressure oxygen (PvO_2) among the three positions. However, PaO_2 was slightly higher in the supine than the lateral positions, though this was not statistically significant.

Good et al., (1979), in an experimental design, compared 24 ventilated post-heart surgery patients with PEEP and without PEEP to determine the efficacy of PEEP in the resolution of atelectasis for this group of patients. The authors reported a high incidence of LLL atelectasis immediately after surgery, and atelectasis persisted regardless of PEEP usage. The theory behind the usage of PEEP is to increase FRC and increase compliance, but in this study PEEP appears not to have altered the incidence of atelectasis. The likely explanation may be that the added volume went to the more compliant area instead of distributing to the low compliance area and opening up the collapsed lung region. There are subtle differences between the two groups. The no PEEP group's PaO_2/PAO_2 ratio showed no significant increase over the course of the 15-hour study period. In contrast, the PEEP group showed significant gradual improvement of this ratio over time. Nevertheless, after extubation, 90% of the patients in both groups developed atelectasis and the degree of atelectasis was not significantly different between the groups.

It is speculated upon and believed by many that large tidal volumes and PEEP will prevent atelectasis, re-expand the collapsed lung region, and improve oxygenation. There is no evidence to support this hypothesis. On the other hand, there are documented studies of the adverse effects of a large tidal volume and PEEP on cardiorespiratory function. Theoretically, positioning has the potential to be equally effective in enhancing oxygenation by indirectly altering the intrapleural pressure and the ventilation/perfusion distribution. Since the existence of inconsistent results concerning positioning and oxygenation among Banasik et al. (1982), Gillespie et at. (1990), Shively (1988), Noll and Fountain (1990), and Banasik (1990), further study is warranted to determine whether oxygenation is affected by positioning using large tidal volumes and low frequency mechanically ventilated post-CABG surgery patients. The findings of such a study may be useful in the post-operative respiratory management of CABG patients.

Positioning and Pulmonary Artery Pressures

The effects of different positions on pulmonary artery pressures (PAP) and pulmonary capillary wedge pressure (PCWP) have been studied by many researchers. Woods and Mansfield (1976) studied 12 outpatients with coronary artery disease. Their findings indicate non-significant variations of PAP and PCWP from flat to 45° backrest position, as long as the transducer is levelled to the phlebostatic axis of the left atrium. Using 32 post-operative CABG patients in their study, Chulay and Miller (1984) evaluated four different backrest elevations (0°, 20°, 30°, 45°) on PAP and PCWP readings. Their findings were identical to those of Wood and Mansfield. Thus, PAP and PCWP are not influenced by backrest elevation so long as the elevation is not more than 45° and adjustment of the transducer to the phlebostatic axis is made.

Kennedy, Bryant, and Crawford (1984) studied the effects of lateral positioning on PAP and PCWP in 25 male patients with various cardiac problems. They found no differences in the pressure readings in lateral positions versus supine positions when the transducer adjustment to the phlebostatic axis was made in the lateral positions. The difficulties in determining an accurate phlebostatic axis while in lateral positions is illustrated by Keating et al., (1986) and Groom, Frisch, and Elliott (1990). In a mixed population of 59 surgical (SICU) and medical (MICU) ICU patients, Groom et al., in a repeated measures design, determined the reproducibility and reliability of two different reference levels for the transducer during lateral positioning. Both units used the intersecting point between the fourth intercostal space and midaxillary line as the reference point in the supine position, and the transducer was mounted on a pole. However, the two units differed in the lateral reference point. The SICU group had the transducer levelled to the fourth intercostal space and dependent midaxillary line; in contrast, the MICU group had the transducer levelled to the fourth intercostal space and sternum. The SICU group resulted in a shift of 1-2 cm of the transducer level on the pole from the supine position, and the MICU group resulted in a 3-5 cm shift on the pole. PAP and wedge pressures (except PA diastolic) of the SICU group were not significantly different in lateral positions. On the other hand, significant differences were noted in all pressures of the MICU group in lateral positions. When the method of levelling was switched between the two units, the findings from this crossover study showed a slight decrease in the differences among the lateral and supine positions of the MICU group and a significant increase in the differences between the lateral positions of the SICU group. This study implies that different groups of patients may require a different anatomical reference for the transducer level in the lateral positions.

Keating et al. (1986), in a small sample of ICU patients, found significant differences in PAP and wedge pressures between supine and lateral positions. This raises a question as to whether the change was due to the reference level or changes in hemodynamics secondary to positioning.

Ross (1988) studied 40 post-operative CABG patients in three different positions (supine, right lateral, and left lateral) with backrest elevation at 20° to determine whether adjustment of the transducer was necessary in lateral positions to obtain a reliable PAP and PCWP. This researcher found that transducer levelled at the supine phlebostatic axis was a reasonable reference point in lateral positions for PAP and PCWP readings. An interesting finding is noted in the study: all subjects had higher PAP readings in the left lateral positions. This raised the question of whether the increase in PAP readings in the left lateral position were related to a reduction in PaO_2 as a result of $\dot{V}/\dot{Q}$ mismatch, as $\dot{V}/\dot{Q}$ mismatching will indirectly influence the PAP. In hypoxemia, the pulmonary circulation will shunt blood away from the area with low oxygen tension; this local HPV response indirectly increases the pulmonary blood volume and pulmonary vascular resistance. Thus changes in PAP in different positions may be related to hypoxemia secondary to $\dot{V}/\dot{Q}$ mismatching.

Positioning and Cardiac Output

There are limited studies available on the effects of postural changes on cardiac output. Grose, Woods, and Laurent (1981) studied the effect of flat and 20° backrest elevation on cardiac output, using the thermodilution method in 30 acutely ill patients. These investigators found no statistically significant association between changes in cardiac output (CO) and the two backrest elevations.

Doering and Dracup (1988) compared cardiac output in 51 post-cardiac surgical patients using the thermodilution method in supine and lateral positions with a backrest elevation of 20°. They found a significant difference in cardiac output among the three positions, especially between the supine and the left lateral positions. The higher cardiac output in side-lying positions was suspected to be related to enhanced preload. Other clinical variables, such as temperature less than 37°C, cardiac index less than 2.3 L/min/m^2, post-operative time less than 12 hours, vasoactive agents, and mechanical ventilation were identified as risk factors for variation in cardiac output with lateral positioning. Consequently, Doering and Dracup suggested cardiac output should be consistently measured in a supine position.

In theory, positioning has the potential to influence cardiac output, since the heart is also a fluid-filled organ. The complexity of press, flow, and resistance relationship in the heart may have minimized any gravitational influences. However, cardiac output is an important factor in oxygenation. Alteration in cardiac output will change the SvO_2, which in turn will influence the calculated shunt. Smith, Cheney, and Winter (1974) utilized healthy dogs and dogs with acid-induced pulmonary edema to determine the contribution of cardiac output to the shunting in three volemic states (normovolemic, hypovolemic, hypervolemic). All dogs were ventilated at 15 ml/kg. The results showed that in healthy dogs during the hypovolemic condition, there was a significant drop in hemodynamic parameters, PvO_2, SvO_2, and shunt with a slight decline in PaO_2; PVR increased significantly. During the hypervolemic condition of these dogs, the opposite of the hypovolemic condition occurred, but the increase was not significant. Similar changes were observed in dogs with pulmonary edema. At the end of the study, veno-veno bypass was performed in the dogs to alter the composition of venous blood returning to the heart. When SvO_2 was increased, there was a significant increase in the shunt in both the healthy and lung-injured dogs. This suggests that SvO_2 has a regulatory role in pulmonary blood distribution, with increased HPV response when SvO_2 is low and diminished response when SvO_2 is high.

Bishop and Cheney (1983) expanded the study of Smith et al. (1974). Twenty-two dogs, healthy and Oleic acid aspirated, had veno-veno bypass performed to determine the effect of FiO_2, PvO_2, and flow on shunt. Keeping the flow constant during room air ventilation of the healthy dogs, an increase in PvO_2 level showed a significant decrease in shunt and a concurrent increase in PaO_2. In the same conditions but the FiO_2 was increased to 1.0, the result showed a significant increase in shunt and PaO_2. Increasing the flow while keeping the PvO_2 constant during room air ventilation increased the shunt significantly, but there were no significant changes during 100% ventilation with the same flow and PvO_2 conditions. In the dogs with diffuse lung injury, during 100% oxygen ventilation when flow was held constant and PvO_2 was increased, shunt and PaO_2 were both increased with no changes in PVR. Increasing the flow but keeping PvO_2 constant in lung-injured dogs during 100% ventilation, shunt and PAP were increased but there were no changes in PVR. Changes in cardiac output or PvO_2 will influence shunting, but the magnitude and direction of change is dependent on FiO_2 and the existence of $\dot{V}/\dot{Q}$ inequality.

The importance of cardiac output as an influential component in arterial oxygen has been demonstrated in many studies (Cheney & Colley, 1980; Kelman, Nunn, Prys-Roberts, & Greenbaum, 1967; Philbin et al., 1970). If hypoxemia is caused by atelectasis, it can be worsened by a low cardiac output. The role of cardiac output can be explained as follows: assuming oxgyen consumption is constant, with a reduction in cardiac output there will be an increase in peripheral oxygen extraction. Subsequently, PvO_2 is lowered and widens the $P(A-a)DO_2$. The lower PaO_2 will further reduce peripheral oxygen availability resulting in further lowering PvO_2, creating a vicious cycle with death as the end.

Clearly there is an interdependency among PaO_2, PvO_2, and cardiac output. If Doering and Dracup's (1988) findings are correct that cardiac output is increased in the lateral positions, it follows that worsening oxygenation can be seen in CABG patients when placed in the left lateral position.

Vasoactive Medications and Pulmonary Gas Exchange

The vasoactive medications that are currently in use for post-CABG are to enhance cardiac contractility, but these drugs may either affect the pulmonary vasculature or interfere with the HPV response.

Sodium Nitropruisside (SNP) is used frequently in the post-operative period to reduce both the preload and the afterload of the heart, but it has been shown to increase intrapulmonary shunting

(Cheney & Colley, 1980). The increase is primarily due to the drug's interfering action with HPV response (Cheney & Colley, 1980; Radermacher, Santak, Becker, & Falke, 1989).

Dopamine affects shunting differently. Dopamine is an inotropic agent; its action is dose-related. Often CABG patients receive a low dosage of this drug for the purpose of enhancing renal perfusion. Low-dose Dopamine can adversely reduce PaO_2, demonstrated by a study done by Rennotte et al. (1989). Ten ventilated patients with respiratory failure were studied using the multiple inert gas method to assess the effect of Dopamine in the $\dot{V}/\dot{Q}$ ratio. They found that with infusion of Dopamine at 5 mcg/kg/min, the shunt increased and PaO_2 decreased significantly from baseline. Perfusion is found to be increased to the zero $\dot{V}/\dot{Q}$ region. The likely mechanism is the augmentation of cardiac output with Dopamine. Other groups of researchers also reported similar findings (Shoemaker et al., 1989; Nomoto & Kawamura, 1989).

Shoemaker et al. (1989) found that with a stepwise increase in Dopamine from 2.5 to 10 mcg/Kg/min, the cardiac index and shunt were increased with a decrease in PaO_2; PvO_2 was unchanged. These changes were not statistically significant.

Nitroglycerin is a systemic veno-vasodilator with specific action in the coronary artery. All post-CABG patients are on a low dosage of this drug during the first period. Its use is to minimize the adverse effect from aortic cross-clamping during surgery and to enhance coronary perfusion. This drug is not known to affect gas exchange. In a study of 21 patients undergoing elective thoracotomy, Nomoto and Kawamura (1989) examined the effects of 1 mcg/kg/min Nitroglycerin, Dopamine, and Dobutamine on gas exchange during one and two lung ventilation. During one lung ventilation, infusion of Nitroglycerin resulted in lowering PaO_2 and increasing shunting; however during two lungs ventilation, only PaO_2 was lowered and shunting was unaffected. Other significant changes during the infusion of this drug were a reduction in cardiac index and the mean arterial pressure. In another study by Radermacher et al. (1989) in which Nitroglycerin was studied in 10 ventilated ARDS patients, the results were similar to previous researches with this drug. They speculate that Nitroglycerin acts in a similar way to Sodium Nitroprusside in that the HPV response is inhibited. Inhibiting the HPV response causes redistribution of blood flow to poorly ventilated or collapsed regions, thus creating $\dot{V}/\dot{Q}$ mismatch, shunt, and a decrease in PaO_2.

Statement of the Problem

The current practice in positioning post-operative CABG patients is turning them from side to side. As discussed earlier, findings by Jansen and McFadden (1986), Wilcox et al. (1988), and Weiland and Walker (1986) note that LLL atelectasis is very prevalent among post-operative CABG patients. The study by Zack et al. (1974) and other studies on unilateral lung disease conclude that positioning the patient on the diseased side results in a decrease in $\dot{V}/\dot{Q}$ ratio and an increased shunt (Remolina et al, 1981; Robichaud, 1990). Hypoxemia can be detrimental to any patient--especially post-operative CABG patients, where cardiac arrhythmias and myocardial infarction may complicate recovery. Banaski et al. (1987) and Banaski (1990) are the only studies so far to examine positioning and oxygenation in post-operative CABG patients. The findings are inconsistent with those of Zack et al. (1974). Another similar study that focuses on positioning and oxygenation in post-operative CABG patients will provide further answers to the question about whether positioning affects oxygenation in this group of patients. For this reason, the focus of this study was on the three different positions that post-operative CABG patients are commonly placed in and the effect on oxygenation as measured by PaO_2 and relative pulmonary shunt.

CHAPTER III

Methods and Procedures

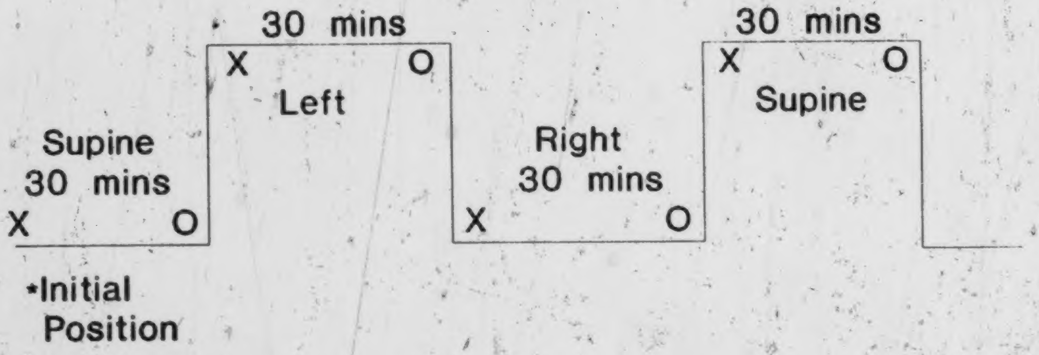
In this chapter a description of the study design, definition of terms, hypotheses, sample, and setting for the study are presented. Ethical considerations associated with the study are briefly addressed. A summary of the pilot study and the resulting changes are discussed, followed by a description of the study protocol. Validity and reliability of the dependent variable measures are highlighted. Finally, the approach to analyze the data is presented.

Research Design

A repeated measures design (Figure 1) was used to investigate the effect of three different positions (supine, right lateral, and left lateral) on PaO_2 and relative pulmonary shunt. The study design allowed each subject to serve as his/her own control. Subjects were exposed to three positions. Counterbalancing was utilized to minimize any potential effect that the order of positional change had on the dependent variables. Six possible sequences (Table 1) were generated by permutation of the three positions. Each subject was randomly assigned to one of the six sequences. The design was chosen to establish which position had the highest PaO_2 and lowest relative shunt for this sample of patients. The independent variables in this study were the three different positions and the dependent variables were PaO_2 and relative shunt.

Figure 1. An example of sequencing schedule

Group VI


Supine
30 mins
X
O
30 mins
X
O
Left
Right
30 mins
X
O
30 mins
X
O
Supine
*Initial
Position

X = Positioning
O = Blood sampling

*The subject's initial position is the position the subject is found to be in at the start of the study.

Table 1

Position Sequencing Schedule

Group	Position Sequence		
I	Right	Left	Supine
II	Right	Supine	Left
III	Supine	Right	Left
IV	Supine	Left	Right
V	Left	Supine	Right
VI	Left	Right	Supine

Definition of Terms

The following terms were operationalized or defined for this study:

Coronary artery bypass graft (CABG) is a surgical revascularization of occluded blood vessel(s) in the heart using grafts taken from either the saphenous vein or the left internal mammary artery. This surgery involves long anaesthetic time and long cardiopulmonary bypass time.

Positioning is the way the body is placed.

Head of the bed elevated (HOB) is a position in which the angle of the headrest is elevated by 20° and one pillow is placed beneath the subject's head. The 20° headrest is measured by an angular ruler. This elevation is the preferred position of most CABG patients to diminish stress on the chest incision.

Supine position is a position in which the subject is placed on his/her back, with one pillow beneath his/her head with HOB elevated.

Right lateral position is a position in which the subject is placed on his/her right side with a 30° hard foam wedge for back support, one pillow between his/her legs, and one pillow beneath his/her head with the HOB elevated.

Left lateral position is a position in which the subject is placed on his/her left side with a 30° hard foam wedge for back support, one pillow between his/her legs, and one pillow beneath his/her head with the HOB elevated.

Oxygenation is a process in which arterialization of blood occurs by coordinating ventilation and perfusion in the lung. It is represented by partial pressures of arterial oxygen (PaO_2) and carbon dioxide ($PaCO_2$). It is measured by arterial blood gases obtained from the arterial line.

Atelectasis is a condition in which a region of the lung becomes airless. The etiology of atelectasis in post-operative CABG may be a summation effect of the anaesthesia, cardiopulmonary bypass (CPB) and surgery. Diagnosis of this condition is based on roentgenogram.

Hypoxemia is a term which refers to low oxygen content in the blood. Three common causes of this condition have been outlined by Shapiro, Harrison, Cane, and Templin (1989). First is ventilation/perfusion inequalities (synonymous to venous admixture, low $\dot{V}/\dot{Q}$ ratio) in which there is more perfusion in relation to ventilation. This $\dot{V}/\dot{Q}$ inequality is the major cause of hypoxemia. The second cause is an increase in zero $\dot{V}/\dot{Q}$ regions (synonymous to refractory hypoxemia). This region is caused by perfusion to totally unventilated alveoli; as a result, venous blood is mixed with oxygenated blood. Increasing FIO_2 in this condition will not correct the hypoxemia. The $\dot{V}/\dot{Q}$ of zero is different from the anatomical shunt, which is also refractory to an increase in FiO_2, but the latter is a normal condition not associated with lung pathology. Normally, anatomical shunt is 3-5% of the cardiac output; this is due to venous blood from bronchial and thebesian veins draining directly to the left atrium. The last condition of hypoxemia is decreased mixed venous oxygen content. In this situation, any factors that may cause an increase in oxygen consumption, a decrease in cardiac output, or a decrease in PaO_2 will result in a decline in mixed venous oxygen content. The drop in mixed venous oxygen content worsens due to the effect of any lung pathology on PaO_2.

Relative pulmonary shunt is a summation of different lung regions where there is perfusion and no ventilation (anatomical shunt), or low ventilation and perfusion ($\dot{V}/\dot{Q}=0$, $\dot{V}/\dot{Q}=.1$). The relative shunt is measured by simultaneous sampling of a mixed venous gas from the distal port of the pulmonary artery (PA) catheter and an arterial gas from the arterial line. The relative pulmonary shunt is calculated based on the formula:

$$(\dot{Q}_{rs}/\dot{Q}_t) = C\acute{c}O_2 - CaO_2/C\acute{c}O_2 - CvO_2$$

CcO_2 is the oxygen content of end capillary blood and is calculated based on the formula:

$CcO_2 = (Hgb \times 1.34 \times S\acute{c}O_2) + (.0031 \times PAO_2)$

CaO_2 is the oxygen content of arterial blood and is calculated based on the formula:

$CaO_2 = (Hgb \times 1.34 \times SaO_2) + (.0031 \times PaO_2)$

CvO_2 is the oxygen content of mixed venous blood and is calculated based on the formula:

$CvO_2 = (Hgb \times 1.34 \times SvO_2) + (.0031 \times PvO_2)$

where: PaO_2 and PvO_2 are the partial pressures of oxygen in arterial and mixed venous blood. $Sc'O_2$, SaO_2 and SvO_2 are percentages of oxygen saturation in end capillary, arterial, and mixed venous blood respectively. Hgb is the hemoglobin value of the patient. PAO_2 is the alveolar partial pressures of oxygen and is calculated based on the alveolar air equation:

$PAO_2 = PIO_2 - (PaCO_2/R)$

For this study, the respiratory quotient (R) was assumed to have a value of 0.8. The partial pressure of inspired oxygen (PIO_2) is calculated using the equation:

$PIO_2 = (P_B - P_{H2O})FiO_2$, in which the atmospheric pressure was assumed to be 700 mmHg (the average for Edmonton), the atmospheric pressure of water is 47 mmHg at 37°C, and FiO_2 is the inspired oxygen (Vij, Babcock, & Magilligan, 1981). The end capillary oxygen saturation based on this equation was assumed to be 100% saturated because PAO_2 was always greater than 150 mmHg in all the subjects.

Fractional inspired oxygen (FiO_2) is the percentage of inspired oxygen expressed as a fraction. The inspired oxygen is analyzed by an oxygen analyzer connected to the ventilator.

Cardiac output is the rate of blood flow in the body. Placement of a pulmonary arterial catheter is required. Measurements are done when the subject is in the supine position. The thermodilution method, using a room temperature injectate of 10cc dextrose and water, is utilized to determine the cardiac output. Injection into the proximal port of the PA catheter occurs at end expiration when respiratory influence is minimal, with each injection occurring in less than four seconds. The average of three to five consecutive measurements is the cardiac output.

Phlebostatic axis is the intersecting point between the fourth intercostal space and the mid-point of the dorsal and ventral surface of the body.

Oxygen tension indices are frequently used to detect arterial oxygen deficiency when a PA catheter is not available to obtain a mixed venous blood sample. The common indices are P(A-a)DO2 and PaO_2/PAO_2 ratio.

Oxygen content indices are determined by the hemoglobin level, the arterial oxygen saturation, and the cardiac output. Once the oxyhemoglobin is saturated, varying the FiO_2 will not significantly alter the saturation level, thus these indices are stable with FiO_2. Arterial-venous oxygen content difference ($C(a-v)DO_2$) and shunt are examples of these indices.

Intermittent mechanical ventilation is a mode of ventilation in which the ventilator provides predetermined breaths per minute and at the same time the patient is permitted to breathe spontaneously. The patient's own breathing is synchronized with the mandatory breath delivered by the ventilator (Vasbinder-Dillon, 1988).

Positive end-expiratory pressure (PEEP) refers to "the existence of an airway pressure above ambient at the end of exhalation, independent of inspiratory dynamics" (Shapiro & Cane, 1990, p. 331).

Fluid balance is the difference between fluid intake and output. Fluid intake includes the amount of crystalloid and colloid given during the operation and the post-operative period to the time of the study. Fluid output includes chest tube drainage, urine, nasal gastric drainage, and hemovac drainage. The calculation for 25% excess in extracellular fluid volume is outlined in Appendix A.

Hypotheses

The assumption of this study is based on the theory that atelectasis will behave as areas of venous admixture. Since pulmonary perfusion is influenced by gravitational forces, it follows that perfusion will increase to the dependent regions of the lung. In mechanically ventilated individuals, gas distribution preferentially goes to the non-dependent region; hence a $\dot{V}/\dot{Q}$ inequality exists. In patients with LLL atelectasis, positioning on their left side would cause an increased perfusion to the atelectatic region with no gas exchange, which would result in a zero $\dot{V}/\dot{Q}$ ratio region, an increase in relative shunt, and a low PaO_2 (Appendix B). Therefore, the following hypotheses were formulated:

1. CABG patients with left lower lobe atelectasis confirmed by chest X-ray would have a significant decrease in PaO_2 and a significant increase in relative shunt when positioned in the left lateral position.
2. CABG patients with left lower lobe atelectasis confirmed by chest X-ray would have a significant improvement in PaO_2 and a significant decrease in relative shunt when positioned in the right lateral and supine positions.
3. CABG patients with no atelectatic changes on chest X-ray would

have no significant changes in PaO_2 and relative shunt in the three different positions.

4. CABG patients with left lower lobe atelectasis confirmed by chest X-ray would have a significant decrease in PaO_2 and a significant increase in relative pulmonary shunt from increased cardiac output (decreased $C(a\text{-}v)DO_2$) in the left lateral position.

5. CABG patients with a fluid balance in excess of 25 percent of the extracellular fluid volume from the operation to the time of the study would have no significant changes in PaO_2 and relative shunt in the three different positions.

Setting

This study was conducted in a 10-bed adult cardiovascular intensive care unit in a major tertiary referral hospital in western Canada. Cardiac surgery is performed in this institution daily, with approximately five to ten elective adult cardiac surgeries weekly. Approximately five to six of these are CABG surgery. All postoperative CABG patients are admitted to this unit directly from the operating room. Once patients are stabilized with a temperature greater than 36.5°C, they are bathed and repositioned into a side-lying position. Turning is part of the routine responsibilities of the nurses, and patients are normally turned every one to two hours. Tracheal instillation and suctioning are done every four hours for all intubated patients. A convenient time to initiate the study was arranged between the researcher and the bedside nurse once stabilization of each patient had been established.

A research assistant was employed to assist with the repositioning of each subject and the transporting of blood gases to the laboratory. All data collection, including blood sampling and cardiac output, was done by the researcher. However, the bedside nurse often voluntarily participated in the turning. In the study, the subject was repositioned a total of three times with an elapsed time of 30 minutes between turns. The majority of the subjects were studied between 1600-2400 hours on the day of surgery. Environmental factors that might affect oxygenation such as the temperature of the room and background noises (alarms, voices, etc.) were not controlled. Background noise in an intensive care unit has been shown to cause anxiety in patients. However, these environmental factors were recorded and taken into account in the data analysis and the interpretation of the results.

After obtaining ethical clearance, the researcher provided an inservice of the study to the staff in the unit. The role of the researcher and research assistant were outlined as well as the expected role of the bedside nurse. Permission letters from all the surgeons and the abstract of the study protocol were given to each of the four cardiovascular residents and the two physician assistants in the unit. Letters were sent to the Respiratory Therapy Department to inform them of the study.

Sample

The target population consisted of adult patients who required elective CABG surgery. A convenience sample of 30 subjects was selected, based on the selection criteria outlined in Appendix C. Criteria were divided into pre-operative and post-operative periods;

patients had to meet these two sets of criteria before they were admitted into the study. Selection criteria were to minimize confounding variables that might influence the dependent variables; the explanation for each is provided in Appendix C.

The normal variation of PaO_2 between different positions in healthy individuals is less than 10 mmHg (Levitzky, Cairo, & Hall, 1990). The effect size for the dependent variables is very small. The number of subjects determined by the power table for a small effect size (d=.20) at alpha (two-tail) of .05 is 200 to achieve a .51 power of the test (Cohen, 1977). The sample size of 200 subjects was beyond the scope of this study. A sample size of 30 was chosen, providing a power of .12 for the study. The rationale for the small sample size was that if clinically important changes in PaO_2 occurred, the small size would likely show this. Also, the time constraint of the researcher and the financial demands of the study were important. However, the within-group design compensated for the small sample size. In spite of the small sample size and the small effect size, there were definite benefits in conducting this study. Others had not evaluated the effects of positioning on shunting for this group of patients. There was also an increase in the control of confounding variables in this study which others had not done. Furthermore, this research might be used as a seminal study for other researchers to replicate for the enhancement of nursing knowledge.

Ethical Considerations

Ethical approval for the study was obtained from the Hospital Ethical Review Committees where the study took place. In addition, approval for access to subjects was obtained from the two cardiovascular surgeons, the Surgeon-in-Chief of Cardiovascular Surgery, the Director of the Cardiovascular Surgical Intensive Care Unit, and the Nurse Manager of the unit.

Potential subjects who met the pre-operative criteria were informed of the study by the Patient Educator Nurse. Interested subjects were asked if their names could be forwarded to the researcher. When the researcher met with the potential subjects, the study was explained to them and an informed consent (Appendix D) was obtained. Potential subjects were informed by the researcher that the frequency of turning was in excess of the standard practice of the unit. They were made aware of the pain that was associated with frequent turning in the early post-operative period. The potential subjects were advised that an analgesic would be given prior to the study and upon completion. Since the subject was ventilated at the 0time of the study and unable to verbally express his/her discomforts or desire to withdraw from the study, the bedside nurse was instructed prior to the study to be the subject's advocate. A copy of the consent was given to each subject as reference.

Each subject was made aware that he/she would not be identified in the reporting of the study. Information from subjects was coded to maintain confidentiality. Only the researcher had access to the subjects' names and the consent forms were kept in a locked drawer. Subjects were made aware that they had the right to refuse to participate in the study without prejudicfng their care.

Pilot Study

The purpose of the pilot study was to evaluate the feasibility of the study protocol and the adequacy of the data collection sheet, the ability of the blood gas laboratory to absorb extra analyses, the training of the research assistant, the problems that might be encountered, and the opportunity to make any revision in the study protocol.

The first pilot subject was a patient who had given consent for the study but failed to meet the post-operative criteria due to intra-operative complications. Nevertheless this patient was studied, but the results were excluded from the final data. The experience with this subject resulted in major revision of the data collection sheet, as this patient's hemodynamic parameters fluctuated widely and this could not be reflected in the original form. There was a problem with the width of the hard foam wedge, as well as the degree of headrest elevation. After the first pilot subject, three patients were approached; two of whom refused because of fear and one consented but had a malfunctioning pulmonary artery (PA) catheter. With the second pilot subject, another problem arose due to the subject's fluctuating temperature. There was a period of a week before the next pilot subject was conducted. No additional problems were encountered, and the revised data collection sheet appeared satisfactory to the researcher. The experiences from the pilot study resulted in modification of the data collection sheet, the size of the hard wedge foam, the headrest elevation, temperature-corrected blood gases, and the selection criteria.

Data Collection Sheet

The data collection sheet was revised so that subjects were monitored in their initial position for half an hour prior to any changes in position. The inclusion of this half hour was to establish that the patient was hemodynamically stable. Furthermore, hemodynamic and respiratory parameters were recorded at 0, 5, 15, and 30 minute intervals to demonstrate the changes in these parameters with turning. The first extubated arterial blood gas was also recorded for comparison. Variables were also added to the data collection sheet such as Albumin in the cardiopulmonary bypass (CPB) prime solution and types of anaesthesia used. The revised data collection sheet is in Appendix E.

Size of the Hard Foam Wedge

The original foam wedge's base spanned three-quarters of the width of the stretcher and difficulties were encountered with its placement. As a result, 5 cm was cut from the base while preserving the 30° angle of the wedge, which eased placement.

Degree of Headrest Elevation

From repeated measurements of volunteer patients and the two pilot subjects, a 20° headrest elevation with one pillow beneath the head was needed, in order to achieve a 30° elevation of the head and 20° elevation of the thorax.

Temperature Corrected Blood Gases

The issue of temperature corrected blood gases arose when the second pilot subject developed a fever. The laboratory was not consistently correcting all blood gas values to the patient's core temperature, so for the purpose of consistency, temperature correction in this study for the blood gases was done for temperatures greater than 38°C or less than 36°C; otherwise all blood gases were measured at 37°C. Temperature correction for hyperthermia increases the PaO_2 value, making it difficult to delineate the cause of the PaO_2 changes (Shapiro et al. 1989).

Sample Selection Criteria

The criteria to limit the age between 50-70 was changed because more surgery was being performed on patients whose age was outside this range. Considering the difficulties in recruitment, the age criterion was changed to anyone over the age of 18 years. The duration of the post-myocardial infarction period was also altered from six months to six weeks. It appears that more patients are being operated on shortly after their myocardial infarction if their occlusions are severe. A recent study has shown that myocardial infarction does not increase surgical mortality rates. In addition, the patient's temperature criterion was dropped because most post-CABG subjects studied had a temperature of 38° or more.

Data Collection Procedures

The data collection procedures (Appendix F) described in the following section include the revisions made from the pilot study. The subjects for this study were referred from the patient educator who saw potential subjects for pre-operative teaching. The researcher phoned the patient educator twice daily to confirm potential subjects chosen from the weekly surgical schedule. The patient educator would then approach those who appeared to have met the pre-operative criteria to see if they would be interested in having the researcher speak to them about the study. If the patient agreed, their name was forwarded to the researcher. The researcher then approached these potential subjects for a voluntary informed consent (Appendix D).

On the day of the subject's surgery, the researcher visited the intensive care unit at 1300-1600 hours to see if the subject who gave consent had returned from surgery. The post-operative criteria (Appendix C) were assessed by the researcher; if the subjects met these criteria, then they were admitted to the study. At the same time, the researcher arranged a convenient time with the bedside nurse to initiate the study.

At the pre-arranged time, the researcher returned to the unit to initiate the study protocol. Generally, initiation of the study protocol started on the operative day four to ten hours after the subject's surgery. The timing for the study was to ensure that all subjects were ventilated. The researcher checked to ensure that all the lines in place from surgery were functioning. Then the headrest 20° elevation was measured by a protractor and one pillow was placed under the subject's head. The PA and arterial line transducers mounted on an intravenous pole were levelled with a carpenter's level to the phlebostatic axis. These transducers, connected to a Hewlett Packard monitor, were recalibrated and zeroed as per unit protocol. Adjustment of the transducer's level was not done for lateral positions (Ross, 1989). The subject's current position and the duration in this position were recorded. The respiratory management for the subjects followed the standard protocol of the unit. Two brands of ventilators were used: Servo 900 and Bear 2. All patients were on intermittent mechanical ventilation mode with individual adjustment for FiO_2 and PEEP. No ventilator changes were made during the study; if changes were necessary, then the study was terminated. Infusion of inotropic and intravenous fluid were recorded along with any changes made during the study. At the start of the protocol, each subject was informed that the researcher was initiating the study and an intravenous analgesic was given prior to turning. Recording of hemodynamic and respiratory variables began, in the position in which the subject was found, for half an hour (0, 5, 15, and 30 minute time intervals) before any blood sampling was carried out. At 30 minutes, simultaneous arterial and mixed venous blood samples were drawn from the arterial and PA catheters in place from surgery. These blood samples were submerged in ice slush immediately. Cardiac output was done using the thermodilution method and an Edwards' cardiac output bedside computer. The cardiac output value recorded was the average of three to five measurements. Cardiac output was done only in the supine position. Lateral positions might have altered this value, but it was impractical to do cardiac outputs in all positions because of the extra fluid putting these subjects at risk of heart failure.

Subjects were randomly assigned to one of the six turning sequences as previously outlined in Table 1. However, every 30 minutes, turning was standardized among all the subjects. In instances where the subject had to be suctioned during the stabilization period, there was a waiting period of 30 minutes from the time of suctioning was done before taking blood samples. The study protocol was then resumed. Subjects were repositioned by the researcher and the research assistant according to the assigned sequencing schedule. However if the subject's initial position was supine and he/she happened to be assigned to group III or IV, the subject was turned to the next position in the sequence and the last position was the supine. The same modification was made to accommodate the subject's initial position if it was a lateral position. In lateral positions, the hard foam wedge was placed behind the subject's back for support and to keep a trunk rotation angle of 30°. A pillow was also placed between the subject's legs. Measurements of cardiopulmonary parameters were obtained at intervals of 0, 5, 15, and 30 minutes. At 30 minutes, arterial and mixed venous blood were drawn simultaneously by the researcher and immersed in ice slush immediately. The blood samples were taken to the blood gas laboratory by the research assistant within five minutes. These steps were repeated until all the positions in the sequence were assumed by the subject. Other variables on the data collection sheet were obtained from the subject's chart.

In the study, a total of three turns, four sets of blood samples and three to five cardiac output measurements were performed on each of the subjects. Upon completion of the study, an analgesic was given to each subject as per physician's order to minimize any possible discomforts from repeated turning.

Validity and Reliability of Measurements

A number of steps were taken to ensure that the values obtained for the dependent variables were valid and reliable. The following discussion encompasses the actions that were taken in the study to assure the validity and reliability of the dependent variables.

Criteria for Determining Pulmonary Abnormalities

The indices of pulmonary function are lung mechanics and oxygenation; the latter is a reflection of the combined effects of the cardiorespiratory system. In clinical settings, PaO_2 is a common criterion for assessing oxygenation and pulmonary dysfunction. The PaO_2 value by itself may not be meaningful; however, when this value is interpreted in context with FiO_2, it may provide some information as to the direction of change without information on the magnitude of change. Consequently, oxygen-tension base indices have been suggested for providing direction and magnitude of oxygenation impairment. The popularity of the oxygen-tension index is its simplicity, because it does not require a mixed venous blood sample. The first oxygen-tension base index is $P(A-a)DO_2$ (Shapiro et al., 1989). Cane, Shapiro, Templin, and Walther (1988) compared different oxygen-tension based indices to oxygen-content base indices in 75 ICU patients. These researchers calculated the shunt based on clinical FiO_2. The study shows that for stable patients on room air, $P(A-a)DO_2$ is a reliable index to estimate the relative shunt. However, the validity of this index changes in critically ill patients with supplemental oxygen. Different FiO_2 will alter the SaO_2 and SvO_2 values and also the $P(A-a)DO_2$. The authors point out that this oxygen-tension base index is the least reliable index for estimating the shunt. In contrast, the a/A ratio is not influenced by FiO_2. This is illustrated by re-arranging the equation (Gilbert & Keighlely, 1974):

$$PaO_2/ PAO_2 = 1 - [P(A-a)DO_2/PAO_2]$$

This corrects for different values of FiO_2. The a/A ratio index is considered a more reliable reflection of shunt (Ahrens & Rutherford, 1987), but Cane et al. (1988) have found that there is a constraint in its reliability. Only at a FiO_2 between .30 and .55 will this ratio show a predictability to shunt at a correlation of -.72.

In 12 post-operative ventilated patients, Herrick, Champion, and Froese (1990) replicated a similar study by Cane et al. (1988), except a repeated measures design was used for five different FiO_2 (.3, .45, .60, .75, 1.0). Their findings are in keeping with those of Cane et al. (1988). Based on these studies, a comparison of $P(A-a)DO_2$ to shunt when patients are on supplemental oxygen is not valid and that a/A ratio is a much better estimate of the shunt with FiO_2 between .3 to .55. These two indices do not provide the true shunt. The differences in their accuracy may be due to assumptions made in the alveolar air equation in which $PaCO_2$ equals $PACO_2$, respiratory ratio equals .8, and PH_2O equals 47 mmHg (Shapiro et al., 1989). Additionally, inconsistency in temperature correction for blood gas values will influence the results of these indices and contribute to the inaccuracy.

The value of temperature correction in blood gases is a controversial topic. Temperature alters the oxygen affinity of hemoglobin and therefore the oxyhemoglobin dissociation. However, the PaO_2 value is raised with temperature correction. This change in PaO_2 value has no relationship to lung function. Shapiro et al. (1989) advocate that blood gases should be measured at 37°C, as temperature corrected PaO_2 values do not reflect the actual oxygen homeostasis. For purposes of consistency in this research, temperature corrections were done for core temperatures greater than 38°C.

For comparison purposes, oxygen-tension indices are included in the results. Oxygen-tension indices and PaO_2 are frequently used to dictate treatment modality, but the standard for quantifying oxygen transfer abnormalities is the shunt. Shunting results in hypoxemia, and there are many conditions that cause shunting. These have been discussed already. The shunt fraction is a calculated value based on both arterial and mixed venous oxygen values. This fraction is considered a true measure of intrapulmonary shunt, with the normal value of 3% to 5% (Cane et al., 1988). The true shunt fraction is calculated when the patient is breathing 100% oxygen; however, it has been shown that an FiO_2 of 1.0 increases the true amount of shunting in the lung (Shapiro et al., 1989; Douglas, Downs, Dannemiller, Hodges, & Munson, 1976).

Suter, Fairley, and Schlobohm (1975) compared the shunt fraction at clinically indicated FiO_2 and FiO_2 at 1.0 in 20 ventilated ICU patients with respiratory failure. They conclude that shunt increases significantly when patients are on 100% oxygen. Significant changes at FiO_2 of 1.0 are an increase in shunt, PaO_2,

$P(A-a)DO_2$ and a decrease in functional residual capacity (FRC) and pulmonary artery pressure with no changes in wedge pressure. The reason for the increase in shunting is that although 100% oxygen eliminates the low $\dot{V}/\dot{Q}$ regions of the lung, resorption atelectasis results from breathing pure oxygen explains the reduction in FRC. Furthermore, hyperoxygenation of mixed venous blood inhibits the hypoxic vasoconstriction response and causes redistribution of blood flow to nonventilated regions which leads to shunting. In summary, when shunt is calculated at 100% oxygen, it is an increase in the true intrapulmonary shunting compared to lower FiO_2. With the addition of PEEP, there is a significant decrease in shunt, $P(A-a)DO_2$, and increase in PaO_2 and FRC, with no changes in PvO_2 regardless of the FiO_2. Suter et al. (1975) speculate that there is recruitment of collapsed alveoli and improvement in $\dot{V}/\dot{Q}$ inequality. The observed changes in FRC and $P(A-a)DO_2$ support the action of PEEP. FRC and the compliance of the lungs are similar at clinical FiO_2 and at a FiO_2 of 1.0 with PEEP, suggesting that PEEP may prevent absorption atelectasis. In a subset analysis, Suter et al. (1975) divide patients into high PCWP and low PCWP groups; comparison of this variable on shunt shows that higher shunting could result from a low FiO_2 and a high PCWP or a high FiO_2 and a high PCWP. The difference in response between the two PCWP groups may be due to changes in the forces governing edema formation and the passive distending force from the high PCWP. In addition, inhibition of the hypoxic pulmonary vasoconstrictor (HPV) response may alter the pressure in the capillaries around the atelectatic area which in turn may lead to loss of protein and fluid to interstitial space and alveoli, especially when there is damage at the endothelial membrane. The study shows the effect of FiO_2, PCWP, and PEEP on shunt.

In another series, Douglas et al., (1976), in a repeated measures study of 30 ventilated surgical patients, determined the effect of different FiO_2 on shunt fraction calculation. In their study, each patient was subjected to FiO_2 of .21 to 1.0 with stepwise increments of .1. When the shunt fraction was plotted against FiO_2, a convex curve was formed. Shunt is maximum at FiO_2 of .21 and drops rapidly at FiO_2 of .3; at FiO_2 between .3 to .7, shunt change is minimal and the shunt fraction starts to ascend when FiO_2 exceeds .7. There are significant differences in shunt between FiO_2 of .21 and 1.0, with shunting highest with room air breathing. The reasons for the observed changes in shunt are provided by Douglas et al.. During room air breathing, areas with low $\dot{V}/\dot{Q}$ ratio will lower the PAO_2 and the end capillary oxygen content less than the values estimated from the alveolar air equation; therefore the calculated shunt is falsely higher than its actual value. With an increase in FiO_2, the oxygen gradient exits between the alveoli and the capillary which leads to better diffusion. Additionally, increasing FiO_2 will increase PAO_2 in the poorly ventilated area, therefore resulting in an increase in arterial oxygen content and a decrease in shunting. The stable shunt at FiO_2 between .3 and .7 may indicate that low $\dot{V}/\dot{Q}$ regions and diffusion abnormalities have been minimized. However, when FiO_2 is increased to more than .7, shunt increases because of absorption atelectasis and blunting of the HPV response. HPV is thought to occur in low $\dot{V}/\dot{Q}$ regions; the alveolar oxygen in these areas equalizes with the oxygen in the pulmonary artery because these alveoli are not receiving air from the airway. When PAO_2 is less than 60 mmHg, local HPV is activated to redirect blood away from these areas. At high FiO_2, PAO_2 and PvO_2 will rise, diminishing the degree of HPV response leading to perfusion to these low $\dot{V}/\dot{Q}$ regions and an increase in shunting.

Douglas et al. (1979) also looked at the influence of PEEP in two groups of patients. The shunt fraction did not change significantly. However when PvO_2 is low, increasing FiO_2 may raise the shunt; with a widening alveolar-mixed venous oxygen gradient, oxygen rapidly diffuses to the capillaries in excess of gas inflow from the airways and results in alveolar collapse. In this study, varied PvO_2 with different FiO_2 did not cause an increase in shunting.

Oliven, Abinader, and Bursztein (1980) replicated a similar study to that of Douglas et al. (1976) except that four different FiO_2 were used in each patient. The results are similar to those of Douglas et al. (1976).

The practice of measuring shunt at FiO_2 of 1.0 may lead to erroneous treatment of a condition that is induced by the measurement rather than changes in the patient's original pathology. Calculating shunt at clinically indicated FiO_2 has more clinical value than at 100% oxygen breathing (Douglas et al., 1979; Cane et al., 1989).

The dependent variables for determining oxygenation in this study were arterial oxygen tension (PaO_2) and relative pulmonary shunting ($\dot{Q}_{rs}/\dot{Q}_t$). Since it is not accurate to compare PaO_2 among patients breathing different FiO_2, two methods were used to circumvent this problem. The first was to analyze PaO_2 with FiO_2 as a covariate. Second was to use oxygen tension base index a/A ratio to eliminate the influence of FiO_2. The shunt calculation was based on the patient's clinically required FiO_2.

Reliability of the Dependent Variable Measures

There are numerous factors which might influence the dependent variables; some precautions were taken to eliminate these factors. These methods are suggested by Shapiro et al. (1989).

All blood gases were drawn using a pre-heparinized plastic syringe. Heparin has a pH of 7.0 (Shapiro et al., 1984), an excess amount of heparin in the syringe might alter the pH, $PaCO_2$, and PaO_2 of the blood gas values. For this study, the amount of heparin in the syringe was 0.05 ml to anticoagulate 0.5 to 1 ml of blood.

Mixed venous blood samples in this study were obtained via the distal port of the pulmonary artery (PA) catheter using a heparinized 3 cc syringe. A discard of 3 cc of blood was done to ensure the blood was a mixed venous sample. Furthermore, the sample was withdrawn slowly to prevent accidental aspiration of the end-capillary blood into the mixed venous blood. The arterial blood sample was drawn from the arterial line. A discard of 2 cc from the arterial line was done before the 0.5 cc of blood sample was taken. The blood samples were then immersed into ice slush. This is vital, since blood contains living cells and metabolism continues. If left at room temperature for too long, the analyzed values might be inaccurate. Metabolism of these cells is stopped by submerging the syringe in ice slush, as studies have demonstrated that at a temperature of 4°C, cellular metabolism ceases. Both of these blood samples were drawn anaerobically. This is important, since any air bubbles in the syringe will alter the pH, $PaCO_2$, and PaO_2 values. As room air has a $PaCO_2$ of zero and PaO_2 of approximately 147 mmHg, equalization of these partial pressures will occur with blood. Consequently, the results would have a lower PaO_2 and a higher $PaCO_2$. To avoid this error in the sampling of blood gases in the study, all blood samples were drawn anaerobically. If an air bubble was present, it was expelled immediately and capped with a dead end stopper. Improper heparinization of syringes, withdrawing of blood samples, and storage of these samples were potential threats to the internal validity and reliability of the study and steps were taken to minimize them.

Hemodynamic variables such as right atrial pressure (RA), pulmonary capillary wedge pressure (PCWP), and cardiac output are influenced by positive pressure ventilation. In order to minimize the effects of ventilation, all PCWP and RA measurements were taken at end expiration. The cardiac output measurement was done using the thermodilution technique. A 10 cc room temperature injectate of dextrose in water was used for the cardiac output measurement. The time required to inject the fluid was less than four seconds, as slow injection would give a low cardiac output. The cardiac output was an average of three to five consecutive measurements. The injection occurred at end expiration; during this time the respiratory interferences were minimal. In positive pressure ventilation, venous return is decreased on inspiration due to compression of large vessels from the increase in intrathoracic pressure.

There might be a potential for a carry-over effect with rapid turning, even though previous researchers suggest frequency of turning will not influence oxygenation (Banasik et al., 1987; Shively, 1988). In addition, an adequate time to draw a blood sample after any changes in ventilator setting or repositioning has been demonstrated by Douglas et al. (1976), Mathews, Neubert, Conover, and Dion (1985), Schuch and Price (1987), Chulay (1988), and Shively (1988) to be a minimum time of 10 minutes. Thirty minutes was chosen as a conservative time for a rest period in each position and to allow equalization of gases.

The analysis of the blood samples were done in the laboratory by trained technicians using a Corning Model 178 blood gas analyzer, which has an internal one point and two point calibration. In addition, the machine was calibrated every eight hours by the technicians. The reliability of the Corning Model 178 blood gas analyzer is 99.4 percent for PaO_2, 99.1 percent for $PaCO_2$, and 100 percent for pH (Metzger et al., 1987). In this study, the same analyzer was used to analyze both the arterial and mixed venous blood samples to enhance reliability.

Estimation of cardiac output in lateral positions was determined by the oxygen content difference between the arterial and mixed venous blood. This estimation is valid only when the oxygen consumption is constant; then the cardiac output is inversely related to the $C(a\text{-}v)DO_2$. Any changes in the SvO_2 will indicate problems in either the cardiac or respiratory system if the metabolic needs of the body are constant.

Since all hemodynamic and respiratory variables were digital displays on monitors or ventilators, another person was not utilized to ensure the rater reliability.

Data Analysis

The nature of the study resulted in the collection of nominal, ordinal, ratio, and interval level data. Measures of central tendency were tabulated for demographic data, pre-operative factors, operative factors, and post-operative factors to describe the sample.

Comparison of PaO_2 posed a problem, since subjects were on differing FiO_2. To circumvent this problem, an oxygen tension index a/A ratio was also calculated. Statistical analysis of variance (ANOVA) was done with FiO_2 as a covariate with PaO_2. On the other hand, the shunt fraction, an oxygen content index, was not affected by FiO_2 between .3 to .7 (Douglas et al., 1976). Therefore, one-way ANOVA was done to determine the treatment and the order effect. Subgroup analysis of variables that might influence the dependent variables was also conducted. One-way ANOVA is considered superior to paired t-tests because the alpha level is held constant at the predetermined level for simultaneous testing in equality of multiple means (Glass & Hopkins, 1984). Glass and Hopkins (1984) explain that the alpha 0.05 for one pair of t-test analysis is truly 0.05, but with multiple t-test comparisons the alpha level increases. In other words, the alpha level increases with an increase in the number of comparison groups.

A Pearson's r correlation was done to determine any significant correlations between possible confounding variables and dependent variables. The statistical analyses were done using the SPSSX statistical program (SPSSX, 1988).

CHAPTER IV

Presentation of Findings

The purpose of the study was to determine if positioning is an important consideration for post-operative coronary artery bypass graft (CABG) patients for optimal gas exchange. In this chapter, a summary of the subject's pre-operative and post-operative characteristics are given. Sequencing effect on the cardiorespiratory parameters will be discussed followed by the results of the positioning effect. The analyses of the relevant independent subgroups on the dependent variables are introduced, then the results of the hypotheses. Finally, correlations are presented for relevant variables.

Description of the Subjects

Fifty-three patients who met the pre-operative criteria were approached about the study; 49 consented and four refused. The main reason for refusing to participate was pre-operative anxiety. Out of the 49 subjects, 19 did not meet the post-operative criteria for the following reasons: no pulmonary artery (PA) catheter (N=5), non-functional P catheter (N=1), hemodynamics instability (N=2), external cardiac assist device (N=7), cancellation of surgery (N=1), and post-operative anxiety (N=3). Therefore, the remaining sample of 30 elective post-operative CABG patients was included in the study. Pre-operative characteristics of the subjects are shown in Table 2.

Table 2

Pre-operative Characteristics of Subjects

General Characteristics

Variable	Mean	SD
Age (yrs)	61.70	9.46
Height (cm)	171.47	6.09
Weight (kg)	83.57	12.64
Body surface area	1.96	0.15
Ejection fraction (%)	56.23	14.00
FEV1/FVC (%)*	81.62	7.71

Medical History

	Number of Subjects	
Variable	Yes	No
Angina	29	1
Hypertension	11	19
Diabetes	2	28
Hypercholesterolemia	6	24
Past myocardial infarction	12	18
Recency of infarction		
> 6 yrs	2	
> 4 yrs & < 6 yrs	2	
> 2 yrs & < 4 yrs	0	
> 6 mos & < 2 yrs	2	
< 6 mos	6	
Past history of stroke	1	29
Smoking history	21	9
Quite smoking	18	3

*FEV1/FVC=Forced expiratory volume in one second/forced vital capacity

The sample consisted of 29 males and one female. Their ages ranged from 43 to 76 years, with a mean age of 61.7 years. Most of the subjects were over their ideal weight for their height: the average weight was 83 kg and the average height was 171 cm. Pre-operative angina was prevalent among the sample; only one subject denied any history of angina but had had a recent myocardial infarction. Hypertension was documented in 11 of the subjects, and hypercholesterolemia was found in six subjects. All six of the subjects with hypercholesterolemia had pre-operative angina, two had hypertension, and one had had a recent stroke. Forty percent of the sample had a history of myocardial infarction; half had had their infarction in less than six months. The cardiac ejection fraction of these subjects ranged from 30% to 81%, with a mean of 56%. The history of cigarette smoking was prevalent among this sample; it varied from a quarter to three packs per day for 10 to 45 years. Eighteen of the smokers quit smoking pre-operatively; abstinence from smoking varied from less than one week to more than 34 years. The pre-operative pulmonary function test, the ratio of forced expiratory volume in one second to forced vital capacity (FEV_1/FVC), ranged from 68% to 95%, with an average of 81%. This average was within normal limits, but two subjects showed mild obstructive lung disease (less than 70%). All but one subject had a normal pre-operative chest X-ray.

Operative Characteristics of the Subjects

The operative characteristics of the subjects are listed in Table 3. There were two types of anaesthetic used: seven subjects had analgesic anaesthetics and 23 had a combination of analgesic and inhalation anaesthetics. The average anaesthetic time for the surgery was less than five hours, but it varied from three to seven hours. The common cardiopulmonary bypass (CPB) time and the aortic cross clamp time were 107 minutes and 59 minutes, respectively.

During the five months of the study, a change in the pump additive occurred. Nine subjects had a combined additive of Albumin and Mannitol in their pump fluid and 21 subjects had Mannitol only. Intra-operative complications were noted in 11 subjects; these were coagulopathy (N-4), difficult intubation (N-1), poor myocardial contractility (N-4), and arrhythmias (N-2).

Table 3

Operative Characteristics of Subjects

Variable	Mean	SD
Anaesthetic time (min)	297.83	49.74
Bypass time (min)	107.47	31.16
Cross-Clamp time (min)	58.80	19.36

The sternotomy approach was used with all the subjects. They received two to five bypass grafts with an average of three bypass grafts. Ninety percent of the subjects had left internal mammary artery (LIMA) grafts and all of the subject had a minimum of one saphenous vein graft. Figure 2 shows the distribution of the type and number of bypass grafts. Left pleural and mediastinal chest tubes occurred in 26 of the subjects, other placement for chest tubes are shown in Figure 3.

Post-operative Characteristics of the Subjects

Some post-operative blood work was modified from pre-operative values (Table 4). Hemodilution in cardiopulmonary bypass (CPB) and blood loss during surgery resulted in a drop of hemoglobin level

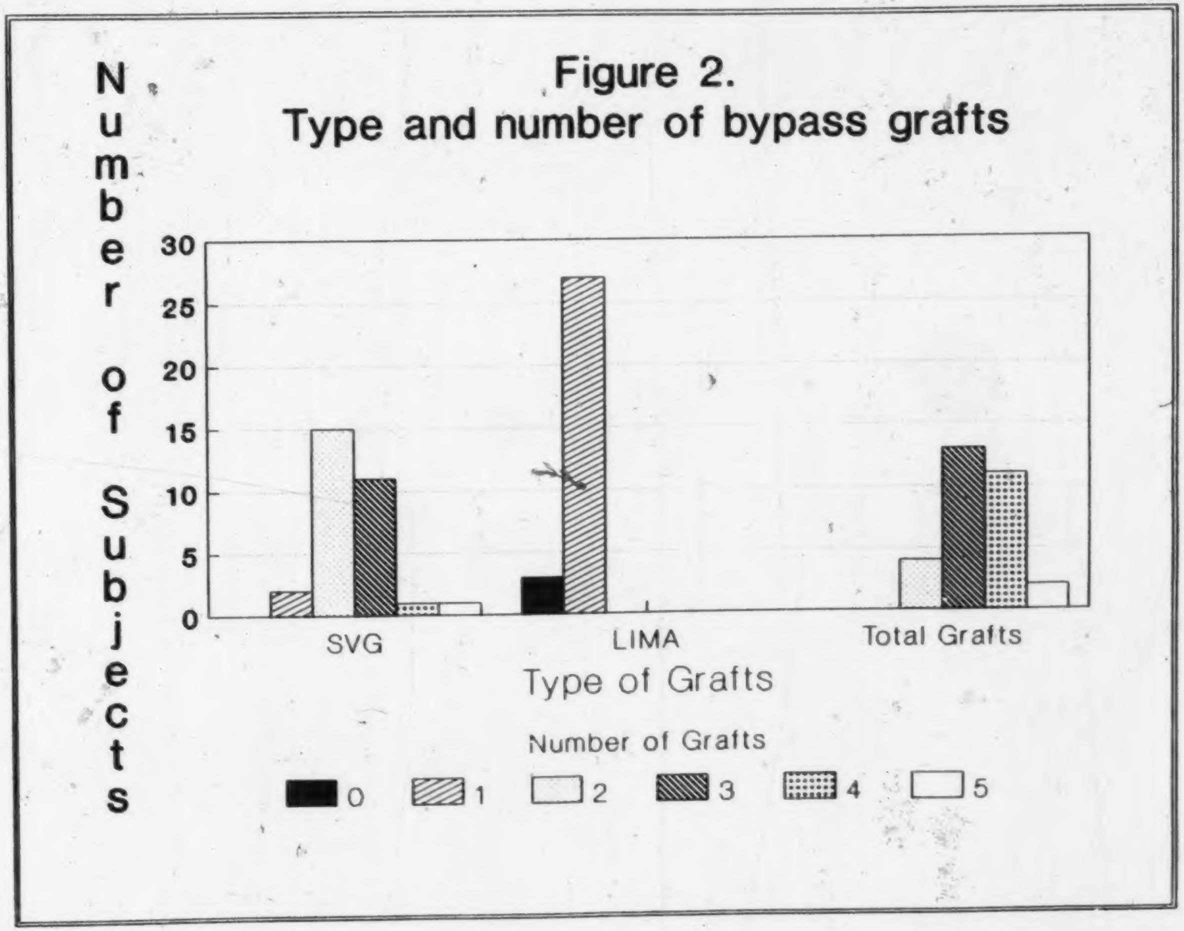
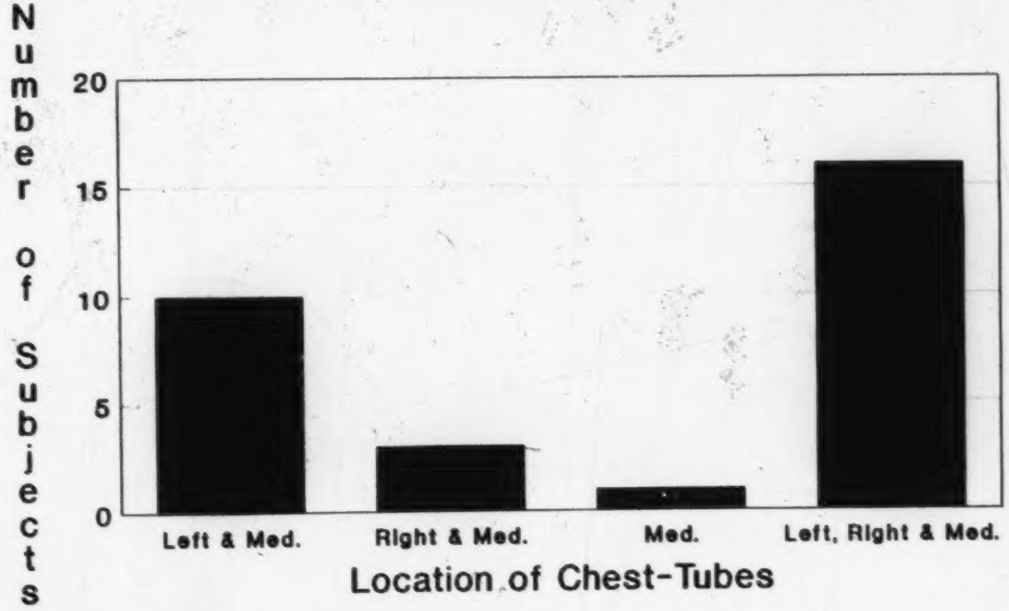


Figure 2.
Type and number of bypass grafts

Figure 3.
Number and location of chest-tubes



from 14.49 mg/dl (*SD*=1.03 mg/dl) pre-operatively to 10.93 mg/dl (*SD*=1.08 mg/dl) post-operatively. This decline persisted despite transfusion in most of the subjects. The serum albumin level was at a low normal level (*M*=31 gm/l, *SD*=4.66 gm/dl) after surgery. In contrast, the serum osmolality level remained within normal limits (*M*=292 mmol/kg, *SD*=6 mmol/kg) despite hemodilution. The mean acid-base balance showed a fully compensated respiratory alkalosis. The average post-operative fluid balance of the subjects was 5.51 liters (*SD*=1.52 L) with an average weight gain of 3.81 kg (*SD*=2.88 kg).

Table 4

Post-operative Laboratory Value Changes of Subjects

Variable	Mean	*SD*
Albumin (g/L)	30.87	4.66
Osmolality (mmol/kg)	291.73	5.59
Hematocrit	0.32	0.03
Pre-operative Hemoglobin (g/dl)	14.49	1.03
Post-operative Hemoglobin (g/dl)	10.93	1.08
Arterial PCO_2 (mmHg)	33.00	5.54
Hydrogen ion	37.00	4.84
Total Serum Carbon dioxide (mmol/L)	22.86	1.76

Subjects were randomly assigned to a turning sequence schedule. The distribution of subjects in the turning sequence schedule was 6,4,1,1,10, and 8 from group one to group six, respectively. The average time to initiate the study protocol post-operatively was 7 hours and 45 minutes and the average time to complete the study was two and a half hours (Table 5). The majority of the subjects were maintained in a supine position from the time of surgery to the initiation of the study period, with an average of 8 hours and 50 minutes (SD-5 hours and 22 minutes) in this position. At the start of the study protocol, there were 28 subjects on Dopamine, 25 on Nitroglycerin (NTG), and 11 on Sodium Nitropursside (SNP). Titration of these drugs was permitted during the study with NTG and SNP being titrated most commonly (Figures 4,5,and 6).

Table 5

Timing of the Study

Variable	Mean	SD
Post-op to Initiation of the Study (min)	465.00	139.47
Duration of the Study (min)	152.60	18.17
Duration in the Supine Position (min)	528.67	321.94
Time of Suction Prior to the Study (min)	96.83	79.31

All subjects were on intermittent mechanical ventilation (IMV). The common IMV rate was 8 breaths per minute and ranged from 6 to 12 breaths per minute. There were two common FiO_2 vales, .3 and .4, (N-7) among the subjects, but it varied from .30 to .75. Positive end expiratory pressure (PEEP) of 5 cmH_2O was usually added, but some patients received a PEEP of 7.5 or 10 cm H_2O (N-10 and N-4, respectively). Tidal volume of the subjects ranged from 850 to 1450 ml with an average of 1242 ml (SD-144 ml) per breath. Table 6 shows the summary of subjects' post-operative respiratory management. All of the subjects were suctioned within four hours prior to the study, and the amount of secretion was classified as small in most subjects except for three, in which large amounts of white sputum were suctioned hourly. Suctioning was done in 30% of the subjects during

Figure 4.
Number of subjects on Dopamine in each position

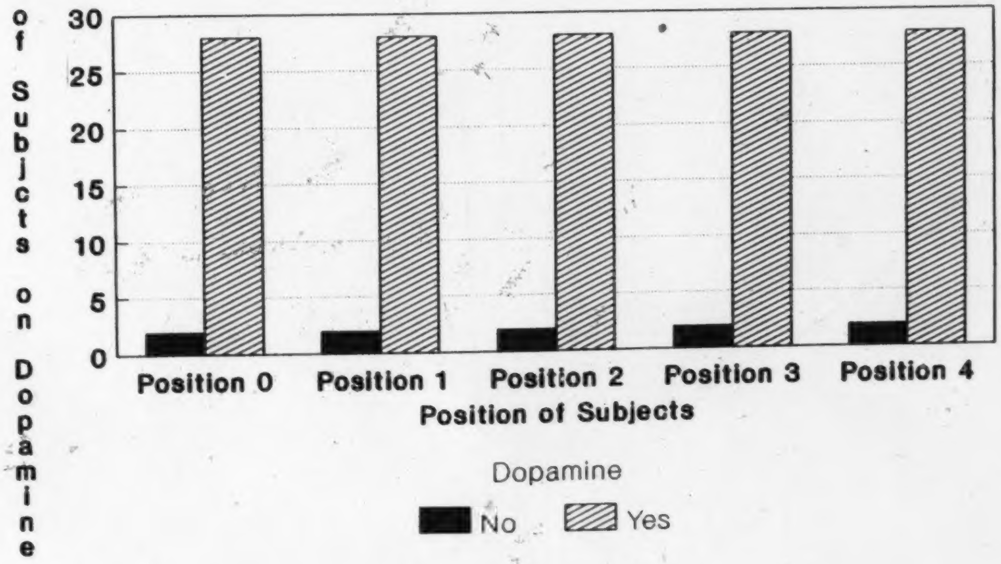
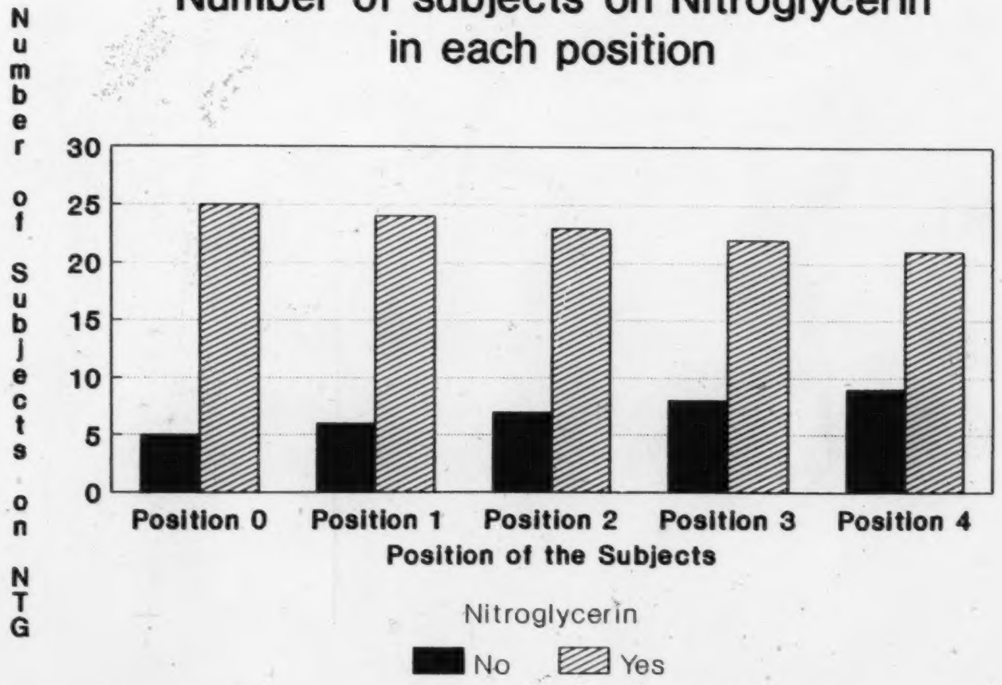
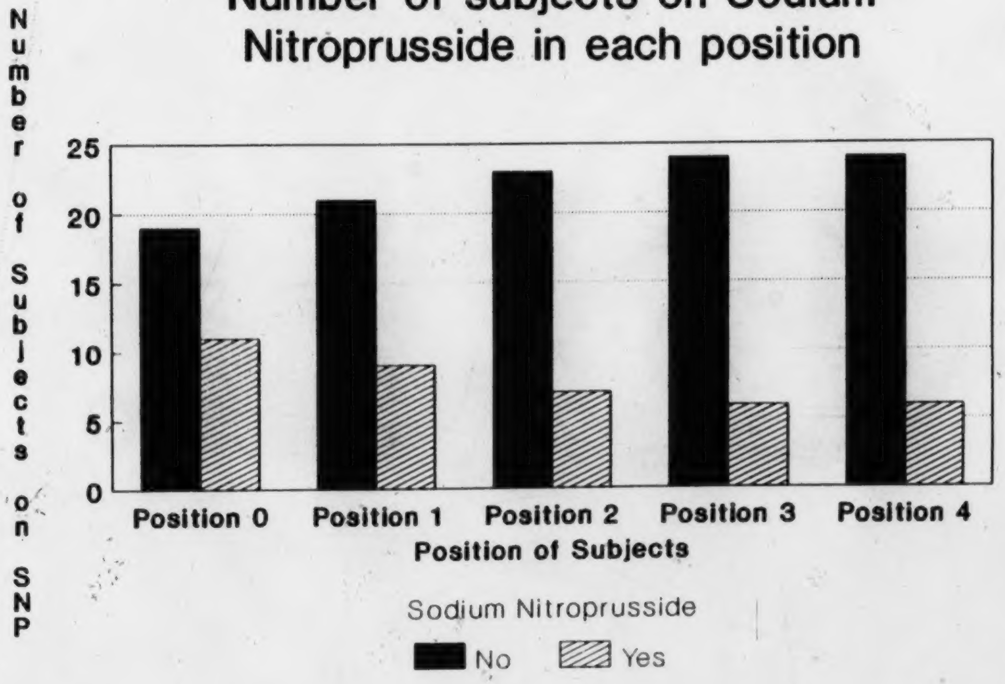


Figure 5.
Number of subjects on Nitroglycerin
in each position



NTG=Nitroglycerin

Figure 6.
Number of subjects on Sodium Nitroprusside in each position



SNP=Sodium Nitroprusside

the study. Most of the subjects were extubated within 24 hours of the surgery; one subject required 48 hours of ventilatory support.

Table 6

Post-operative Ventilator Settings of Subjects

Setting	Mean	SD
Rate	8.27	1.87
Tidal Volume (ml)	242.13	144.22
PEEP (cmH_2O)	6.50	1.81
Inspiratory oxygen (FiO_2)	0.44	0.13

Immediate post-operative chest X-rays did not differ from the chest x-rays taken on post-operative day one. An abnormal X-ray report in the immediate post-operative period was found in 27 of the subjects (Table 7). These reports were from radiologists who were not aware of the study. Quantification of the severity of atelectasis in each subject could not be extrapolated from the X-ray reports. Progressive deterioration in the chest X-ray was documented in all these subjects on day two and day three (Table 7).

Effect of Sequencing on Cardiorespiratory Parameters

One-way analysis of variance (ANOVA) on the difference in hemodynamic and respiratory parameters among the six turning sequences was done to determine the effect of the order of positioning. It was assumed that the order in which the subject was turned had no influence on the dependent variables (Shively, 1988). Sequencing did not demonstrate any significant differences in the parameters tested, except in the heart rate. The significance found

Table 7

Post-operative Chest X-ray Findings

X-ray Findings	Day 0	Day 1	Day 2	Day 3
	Number of Subjects			
Normal	3	3	1	0
*RLL atelectasis (AT)	1	1	1	1
*LLL atelectasis	5	5	1	0
Bilateral atelectasis	21	21	21	4
Bilateral pleural effusions (PE)	0	0	0	3
Bilateral AT & PE	0	0	2	13
LLL PE	0	0	0	1
LLL AT & Bilateral PE	0	0	1	3
RLL AT & Bilateral PE	0	0	0	1
LLL AT & PE	0	0	1	0
Bilateral AT & LLL PE	0	0	1	2
Missing reports	0	0	1	2

*LLL-Left lower lobe; RLL-Right lower lobe.

in the heart rate parameter was subjected to contrast analysis. The result indicated that the main contribution to the significance was in sequence four; one person was in that group. When that individual was eliminated from the analysis, there was no significant difference among the sequences. This indicated that the order in which the subjects were turned had no influence on the hemodynamic and respiratory variables. However, the raw data revealed that all subjects had an increase in cardiorespiratory parameters with turning; this transient increase lasted for five minutes only and this was not reflected in the overall analysis. A summary of the one-way analysis of variance of the sequencing effect on the cardiorespiratory variables is found in Table 8.

Effect of Sequencing on PaO_2 and Relative Shunt

One-way ANOVA was conducted to determine if the ordering of positions had any effect on the main dependent variables. No significant differences in PaO_2 (F=.78, DF=5,24, p=.57) or shunt (F=.14, DF=5,24, p=.89) were shown as a result of the sequencing effect. Furthermore, there was no evidence to indicate that the time lapse between surgery and the time of the study contributed to changes in these dependent variables. This implies that the subjects studied at six hours post-surgery (time one) did not significantly differ from those studied at 10 hours post-surgery (time two). The inclusion of a duplicate position in the design allowed differentiation of changes either due to pathology or due to the treatment effect. There were no changes in mean values between time one and time two with similar positions, which indicates that changes

Table 8

One-way Analysis of Variance of the Sequencing Effect on Cardiorespiratory Variables

Heart Rate

Position	Mean (beats/min) Sequence						DF	SS	MS	F Ratio	P
	1	2	3	4	5	6					
1	92	105	114	55	89	92	5	2616.26	523.25	3.15	*.0252
							24	3988.71	166.20		
2	92	103	114	57	88	91	5	2343.74	468.75	3.11	*.0266
							24	3621.06	150.88		
3	89	99	124	56	89	92	5	2650.03	530.00	3.37	*.0190
							24	3771.98	157.17		
4	88	93	110	59	92	91	5	1454.32	290.86	2.04	.1090
							24	3423.15	142.63		

Systolic Blood Pressure

Position	Mean (mmHg) Sequence						DF	SS	MS	F Ratio	P
	1	2	3	4	5	6					
1	116	106	113	145	115	125	5	1787.52	357.50	1.85	.1412
							24	4640.00	193.33		
2	118	108	127	153	115	129	5	2660.03	532.01	2.69	*.0459
							24	4755.33	198.14		
3	125	106	119	151	115	121	5	2080.62	416.12	1.91	.1298
							24	5226.75	217.78		
4	118	110	120	142	108	123	5	1754.88	350.98	1.69	.1764
							24	4996.48	208.19		

Diastolic Blood Pressure

Position	Mean (mmHg) Sequence						DF	SS	MS	F Ratio	P
	1	2	3	4	5	6					
1	55	61	53	53	55	54	5	133.13	26.63	0.94	.4748
							24	681.83	28.41		
2	59	61	54	55	59	58	5	77.28	15.46	0.79	.5700
							24	472.08	19.67		
3	61	59	57	59	55	58	5	136.77	27.35	0.74	.6044
							24	893.10	37.21		
4	56	63	61	59	56	56	5	217.25	43.45	1.22	.3302
							24	854.75	35.61		

Table 8 Con't

Mean Arterial Blood Pressure

Position	Mean (mmHg) Sequence						DF	SS	MS	F Ratio	P
	1	2	3	4	5	6					
1	71	73	68	72	72	72	5	27.11	5.42	0.17	.9710
							24	762.36	31.77		
2	75	76	73	77	75	77	5	27.13	5.43	0.18	.9670
							24	718.73	29.95		
3	79	72	75	79	72	75	5	185.24	37.05	0.90	.4973
							24	988.23	41.18		
4	73	77	75	76	71	73	5	102.79	20.56	0.54	.7413
							24	907.38	37.81		

Systolic Pulmonary Arterial Pressure

Position	Mean (mmHg) Sequence						DF	SS	MS	F Ratio	P
	1	2	3	4	5	6					
1	27	27	24	34	26	26	5	70.47	14.09	1.42	.2540
							24	238.73	9.95		
2	31	29	25	34	31	29	5	61.59	12.32	0.55	.7396
							24	541.38	22.56		
3	31	25	27	32	27	28	5	100.37	20.07	1.28	.3038
							24	375.50	15.65		
4	27	29	27	32	29	24	5	133.48	26.70	1.78	.1544
							24	359.23	14.97		

Diastolic Pulmonary Arterial Pressure

Position	Mean (mmHg) Sequence						DF	SS	MS	F Ratio	P
	1	2	3	4	5	6					
1	17	17	15	16	17	16	5	8.59	1.72	0.21	.9565
							24	199.71	8.32		
2	19	19	17	16	17	17	5	22.39	4.48	0.71	.6247
							24	152.28	6.34		
3	18	16	18	16	15	16	5	38.51	7.70	1.15	.3641
							24	161.36	6.72		
4	16	19	18	17	17	15	5	49.13	9.83	1.51	.2241
							24	156.23	6.51		

Mean Pulmonary Arterial Pressure

Position	Mean (mmHg) Sequence						DF	SS	MS	F Ratio	P
	1	2	3	4	5	6					
1	21	21	19	22	21	20	5	8.85	1.77	0.26	.9329
							24	166.35	6.93		
2	24	23	21	22	23	22	5	15.97	3.19	0.44	.8137
							24	172.83	7.20		
3	24	20	23	21	20	21	5	68.31	13.66	1.75	.1620
							24	187.56	7.81		
4	21	23	22	22	22	19	5	73.24	14.65	1.97	.1194
							24	178.23	7.43		

Table 8 Con't

Pulmonary Capillary Wedge Pressure

Position	Mean (mmHg) Sequence						DF	SS	MS	F Ratio	P
	1	2	3	4	5	6					
1	13	13	11	14	12	11	5	20.38	4.08	0.81	.5599
							23	116.93	5.08		
2	13	14	11	15	13	12	5	19.39	3.88	0.78	.5749
							23	114.47	4.98		
3	14	12	12	13	11	12	5	34.55	6.91	1.34	.2833
							22	113.16	5.14		
4	13	13	16	15	14	11	5	64.78	12.96	1.50	.2294
							23	199.01	8.65		

Right Atrial Pressure

Position	Mean (mmHg) Sequence						DF	SS	MS	F Ratio	P
	1	2	3	4	5	6					
1	12	13	8	13	12	12	5	25.65	5.13	0.77	.5821
							24	160.35	6.68		
2	14	14	12	13	13	12	5	13.81	2.76	0.37	.8647
							24	179.56	7.48		
3	14	12	13	13	12	13	5	31.02	6.20	1.11	.3827
							24	134.48	5.60		
4	13	15	14	14	13	11	5	38.03	7.61	0.89	.5024
							24	204.78	8.53		

Respiratory Rate

Position	Mean (breaths/min) Sequence						DF	SS	MS	F Ratio	P
	1	2	3	4	5	6					
1	12	9	8	6	9	9	5	73.38	14.68	1.91	.1294
							24	184.08	7.67		
2	13	10	8	8	10	10	5	47.64	9.53	1.02	.4264
							24	223.56	9.31		
3	12	10	8	8	10	10	5	43.24	8.65	1.49	.2306
							24	139.46	5.81		
4	12	10	8	6	11	10	5	40.33	8.07	0.84	.5331
							24	229.83	9.58		

Tidal Volume

Position	Mean (ml) Sequence						DF	SS	MS	F Ratio	P
	1	2	3	4	5	6					
1	1210	1168	1420	1170	1377	1270	5	206589.55	41317.91	1.78	.1546
							24	556323.65	23180.15		
2	1258	1153	1408	1159	1332	1274	5	124095.34	24819.07	1.09	.3928
							24	548004.13	22833.51		
3	1262	1141	1468	1170	1358	1272	5	186904.64	37380.93	1.53	.2181
							24	586569.23	24440.38		
4	1265	1146	1413	1170	1342	1278	5	142074.93	28414.95	1.06	.4059
							24	642518.23	26771.59		

Table 8 Con't

Peak Airway Pressure

Position	Mean (cmH_2O) Sequence						DF	SS	MS	F Ratio	P
	1	2	3	4	5	6					
1	36	.35	42	36	39	36	5	88.60	17.72	0.45	.8103
							24	948.68	39.53		
2	37	37	41	37	41	39	5	82.45	16.49	0.44	.8188
							24	907.14	37.80		
3	37	35	42	37	39	37	5	63.86	12.77	0.30	.9088
							24	1026.36	42.77		
4	35	37	40	37	39	37	5	68.84	13.77	0.32	.8973
							24	1039.80	43.32		

Minute Ventilation

Position	Mean (L/min) Sequence						DF	SS	MS	F Ratio	P
	1	2	3	4	5	6					
1	11.2	10.1	11.4	7.2	11.2	11.8	5	23.50	4.70	0.65	.6661
							24	174.19	7.26		
2	11.5	10.3	11.4	7.3	11.6	12.0	5	25.15	5.03	0.84	.5334
							24	143.40	6.00		
3	11.4	10.2	11.5	8.1	11.6	12.1	5	20.47	4.09	0.66	.6588
							24	149.43	6.23		
4	11.8	10.4	11.5	8.1	11.8	11.9	5	19.00	3.80	0.57	.7203
							24	159.26	6.64		

Arterial Partial Pressure Oxygen

Position	Mean (mmHg) Sequence						DF	SS	MS	F Ratio	P
	1	2	3	4	5	6					
1	128	120	100	98	131	137	5	2601.07	520.21	0.66	.6552
							24	18841.73	785.07		
2	131	117	102	93	125	132	5	2410.41	482.08	0.79	.5646
							24	14570.96	607.12		
3	133	133	100	90	137	143	5	3887.69	777.54	0.92	.4839
							24	20241.78	843.41		
4	130	128	93	91	125	133	5	2822.44	564.49	0.78	.5764
							24	17453.06	727.21		

Arterial Partial Pressure Carbon Dioxide

Position	Mean (mmHg) Sequence						DF	SS	MS	F Ratio	P
	1	2	3	4	5	6					
1	34	37	37	38	33	33	5	67.74	13.55	0.39	.8480
							24	825.23	34.38		
2	33	33	35	38	34	33	5	31.39	6.28	0.24	.9430
							24	639.81	26.66		
3	34	32	37	37	32	33	5	48.09	9.62	0.27	.9225
							24	840.61	35.02		
4	30	34	37	37	32	31	5	93.91	18.78	0.75	.5942
							24	601.06	25.04		

Table 8 Con't

Arterial Oxygen Saturation

	Mean (%) Sequence						DF	SS	MS	F Ratio	P
Position	1	2	3	4	5	6					
1	98	98	98	97	98	99	5	3.28	0.66	0.63	.6799
							24	25.03	1.04		
2	98	98	98	97	98	99	5	3.48	0.70	1.08	.3964
							24	15.48	0.65		
3	99	99	97	97	98	99	5	5.32	1.06	2.51	.0576
							24	10.15	0.42		
4	99	99	97	97	98	98	5	4.44	0.89	2.09	.1025
							24	10.23	0.43		

Venous Oxygen Saturation

	Mean (%) Sequence						DF	SS	MS	F Ratio	P
Position	1	2	3	4	5	6					
1	63	69	70	63	64	68	5	174.23	34.85	0.98	.4484
							24	850.73	35.45		
2	63	65	69	62	66	69	5	180.26	36.05	1.15	.3614
							24	751.61	31.32		
3	65	68	67	60	67	68	5	99.68	19.94	0.99	.4448
							24	483.53	20.15		
4	63	68	68	59	65	69	5	188.38	37.68	1.65	.1809
							24	548.98	22.87		

Relative Shunt

	Mean (%) Sequence						DF	SS	MS	F Ratio	P
Position	1	2	3	4	5	6					
1	10.8	12.8	9.4	11.1	11.1	11.6	5	15.07	3.01	0.13	.9842
							24	558.67	23.28		
2	10.2	11.6	9.1	11.1	11.7	12.1	5	18.34	3.67	0.27	.9241
							24	323.91	13.50		
3	9.7	10.6	11.4	10.8	10.9	11.1	5	8.47	1.70	0.10	.9912
							24	406.56	16.94		
4	9.3	10.7	12.1	10.5	11.8	12.8	5	46.46	9.29	0.64	.6736
							24	350.05	14.59		

*probability ≤ 0.05

which occurred over time were due to the treatment effect (Table 8). Additionally, the process of repeated turning did not demonstrate a consistent increase or decrease in PaO_2 or in relative shunt. This suggests that if there were persistent changes, then these changes could be a result of the treatment. Figures 7 and 8 contain the analysis of the sequencing effect on the dependent variables.

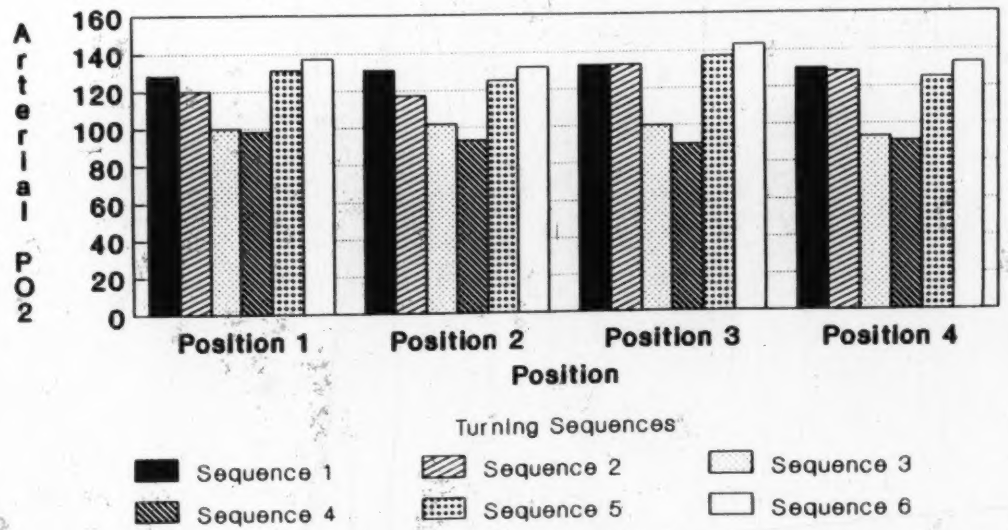
Effect of Positioning on Cardiorespiratory Parameters

The data were reorganized into specific positions (supine, right lateral, and left lateral), and one-way ANOVA was conducted to determine the treatment effect in the cardiorespiratory parameters (Table 9). Significant differences in diastolic blood pressure (F=3.50, DF=4,145, p=.0093), mean arterial blood pressure (F=2.68, DF=4,145, p=.0340), systolic pulmonary arterial pressure (F=6.27, DF=4,145, p=.0001), diastolic pulmonary arterial pressure (F=3.66, DF=4,145, p=.0072), and mean pulmonary arterial pressure (F=7.09, DF=4,145, p<.0001) were exhibited with different positions. The mean values of these parameters were highest in the left lateral position and lowest in the supine position (Table 9). This indicated that the hemodynamic parameters fluctuated in different positions, with most changes occurring in the left lateral position. Table 9 shows the summary of the treatment effect on the cardiorespiratory variables.

Effect of Positioning on PaO_2 and Relative Shunt

The PaO_2 and relative shunt were analyzed using one-way ANOVA to determine the significance of different positioning. No significant changes in relative shunt, oxygen content differences in arterial and venous blood ($C[a-v]DO_2$), and PaO_2 resulted from the

Figure 7.
Sequencing effect and arterial partial pressure oxygen



PO2=partial pressure oxygen (mmHg)

Figure 8.
Sequencing effect and relative shunt

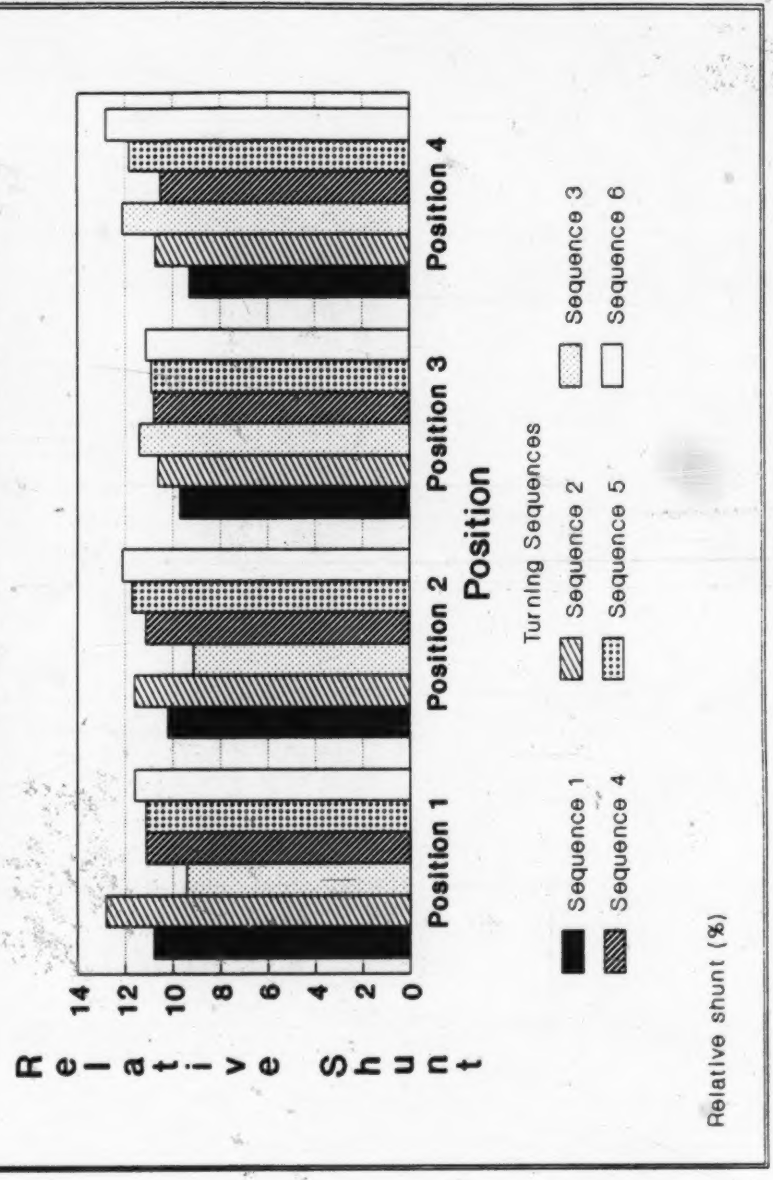


Table 9

One-way Analysis of Variance of the Positioning Effect on Cardiorespiratory Variables

Heart Rate

Mean (beats/min) Position					DF	SS	MS	F Ratio	P
S1	S2	SM	R	L					
92	90	92	93	89	4	282.91	70.73	0.35	.8447
					145	29417.77	202.88		

Systolic Blood Pressure

Mean (mmHg) Position					DF	SS	MS	F Ratio	P
S1	S2	SM	R	L					
118	117	119	115	121	4	559.57	139.83	0.61	.6535
					145	3056.00	227.97		

Diastolic Blood Pressure

Mean (mmHg) Position					DF	SS	MS	F Ratio	P
S1	S2	SM	R	L					
55	56	56	58	60	4	403.11	100.78	3.50	*.0093
					145	4173.77	28.81		

Mean Arterial Blood Pressure

Mean (mmHg) Position					DF	SS	MS	F Ratio	P
S1	S2	SM	R	L					
72	73	73	74	76	4	314.03	78.51	2.68	*.0340
					145	4247.23	29.29		

Systolic Pulmonary Arterial Pressure

Mean (mmHg) Position					DF	SS	MS	F Ratio	P
S1	S2	SM	R	L					
26	26	27	29	30	4	359.53	89.88	6.27	*.0001
					145	2077.80	14.33		

Diastolic Pulmonary Arterial Pressure

Mean (mmHg) Position					DF	SS	MS	F Ratio	P
S1	S2	SM	R	L					
16	15	16	17	18	4	91.89	22.97	3.66	*.0072
					145	910.80	6.28		

Table 9 Con't

Mean Pulmonary Arterial Pressure

Mean (mmHg) Position					DF	SS	MS	F Ratio	P
S1	S2	SM	R	L					
21	20	20	22	23	4	186.84	46.71	7.09	*.0000
					145	955.00	6.59		

Pulmonary Capillary Wedge Pressure

Mean (mmHg) Position					DF	SS	MS	F Ratio	P
S1	S2	SM	R	L					
12	12	12	13	13	4	46.34	11.58	2.09	.0858
					139	771.64	5.55		

Right Atrial Pressure

Mean (mmHg) Position					DF	SS	MS	F Ratio	P
S1	S2	SM	R	L					
12	12	12	13	13	4	56.69	14.17	2.35	.0574
					145	876.27	6.07		

Respiratory Rate

Mean (breath/min) Position					DF	SS	MS	F Ratio	P
S1	S2	SM	R	L					
9	10	10	11	10	4	21.80	5.45	0.67	.6157
					145	1184.20	8.17		

Tidal Volume

Mean (ml) Position					DF	SS	MS	F Ratio	P
S1	S2	SM	R	L					
1282	1275	1275	1280	1275	4	1127.29	281.82	0.01	.9998
					145	3687312.50	25429.74		

Peak Airway Pressure

Mean (cmH_2O) Position					DF	SS	MS	F Ratio	P
S1	S2	SM	R	L					
37	37	37	38	39	4	84.83	21.21	0.59	.6705
					145	5212.88	35.95		

Table 9 Con't

Minute Ventilation

Mean (L/min) Position					DF	SS	MS	F Ratio	P
S1	S2	SM	R	L					
11.1	11.40	11.25	11.45	11.37	4	2.30	0.57	0.09	.9848
					145	902.08	6.22		

Relative Shunt

Mean (%) Position					DF	SS	MS	F Ratio	P
S1	S2	SM	R	L					
11.3	11.1	11.2	11.2	11.2	4	0.90	0.22	0.02	.9995
					145	2123.78	14.64		

Arterial-Venous Oxygen Content Difference

Mean (ml O_2/100ml) Position					DF	SS	MS	F Ratio	P
S1	S2	SM	R	L					
5.08	5.01	5.05	5.08	4.93	4	0.47	0.12	0.12	.9555
					145	102.72	0.71		

Arterial Partial Pressure Oxygen

Mean (mmHg) Position					DF	SS	MS	F Ratio	P
S1	S2	SM	R	L					
128	131	130	128	127	4	312.23	78.06	0.11	.9792
					145	103903.93	716.58		

*probability ≤ 0.05

three positions. The non-significant findings might have been due to FiO_2 being a confounding variable. Analysis of covariance (ANCOVA) on PaO_2 was analyzed with FiO_2 as a covariate to determine if there were any difference among the three positions. The result showed that FiO_2 significantly altered the PaO_2 value (F-43.49, DF-1, p<.001). The treatment effect after removal of the FiO_2 factor remained non-significant (F-.141, DF-4, p-.967) (Table 10). This suggests that although the FiO_2 influences the PaO_2 value, in a within group design the FiO_2 is a constant variable; therefore the decision to either reject or accept the hypotheses from ANOVA and ANCOVA should be similar. The FiO_2 did not change the treatment effect. The lack of change in the dependent variables in the two identical positions indicates that no measurable pathology occurred during the time of the study; the observed change in the dependent variables was because of the treatment effect as previously shown in Table 9.

The mean value for relative shunt in all three positions was identical (M-11.2). A slight difference in PaO_2 among the three positions was noted, with the highest mean value in the supine position (M-130 mmHg) and the lowest in the left lateral position (M-127 mmHg). The right lateral position had an intermediate mean value (M-128). Figures 9 and 10 shows the treatment effect on these two dependent variables.

Analyses of the Subgroups

There was a significant difference between the pre-

Table 10

Analysis of Covariance of the Positioning Effect on Arterial Partial Pressure Oxygen with Inspiratory Oxygen as a Covariate

Source of Variation	Sum of Squares	DF	Mean Squares	F Ratio	P
Covariates	24102.69	1	24102.69	43.49	*.000
FIO_2	24102.69	1	24102.69	43.49	*.000
Main Effects	312.23	4	78.06	0.14	.967
GR	312.23	4	78.06	0.14	.967
Explained	24414.92	5	4882.98	8.81	*.000
Residual	79801.24	144	554.18		
Total	104216.16	149	699.44		

Covariate	Raw Regression Coefficient
FIO_2	102.97

Gran Mean = 128.84

Variable + Category	N	Unadjusted Dev'n	Eta	Adjusted for Independents + Covariates Dev'n	Beta
Gr					
1 First Supine	30	-0.64		-0.64	
2 Second Supine	30	2.09		2.09	
3 Mean of Two Supine	30	1.06		1.06	
4 Right Lateral	30	-0.44		-0.44	
5 Left Later	30	-2.07		-2.07	
			0.05		0.05
Multiple R Squared					0.234
Multiple R					0.484

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*probability ≤ 0.05

Figure 9.
Treatment effect and relative shunt

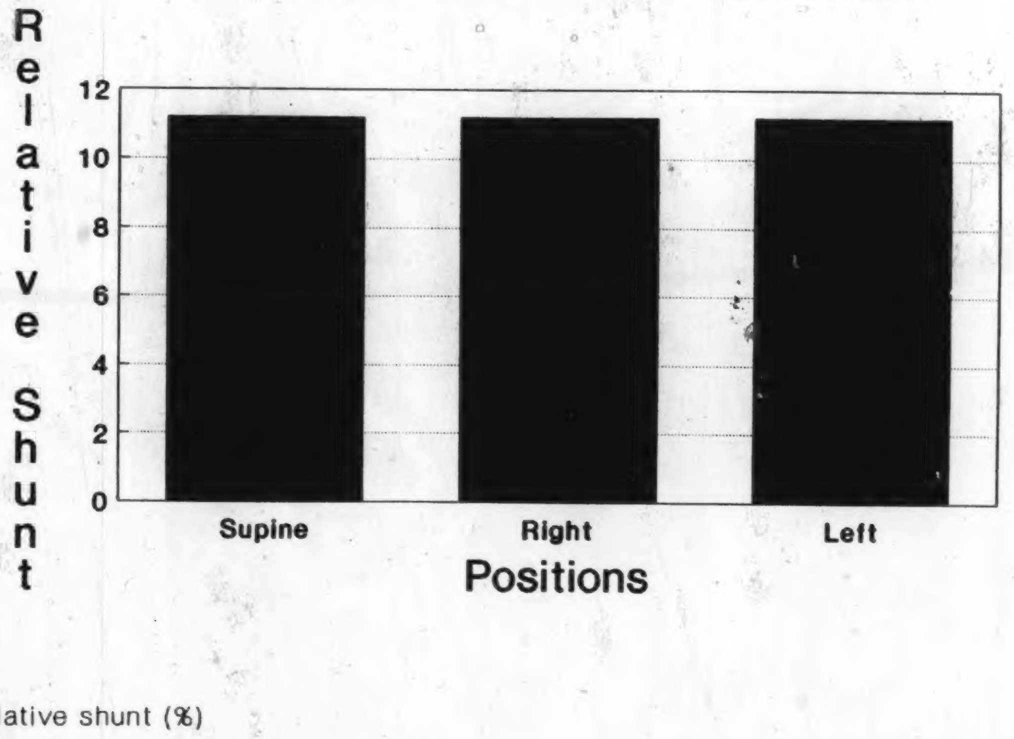
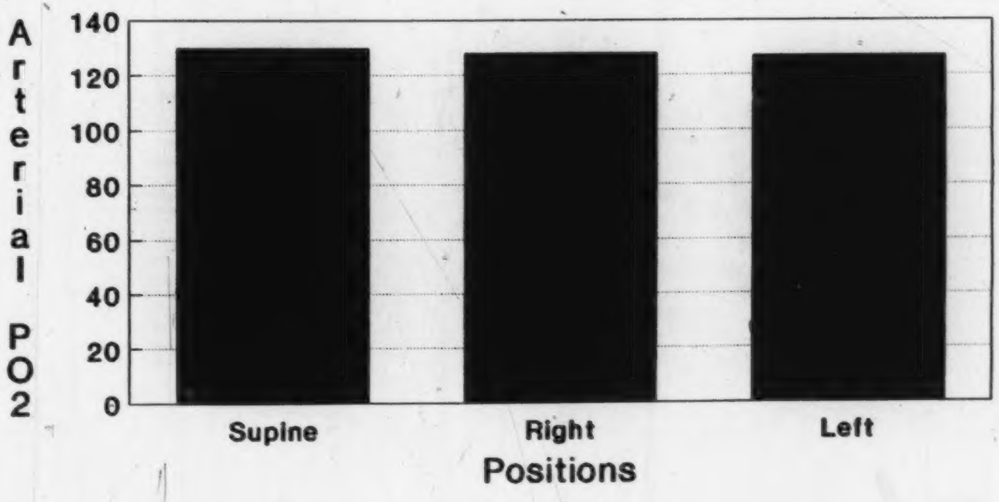


Figure 10.
Treatment effect and arterial partial pressure oxygen



PO2=partial pressure oxygen (mmHg)

operative, post-operative intubated, and post-operative extubation arterial-alveolar ratio (PaO_2/PAO_2) (F=115.23, DF=2,87) suggesting that there was a significant deterioration in ventilation/perfusion after the surgery (Table 11). Contrast analysis showed that the difference was between the pre-operative (M=.812) and both post-operative and extubation (M=.458) a/A ratio. The difference between the post-operative intubated (M=.526) and post-operative extubated a/A ratio (M=.458) was non-significant.

Table 11

One-way Analysis of Variance of Pre-operative, Post-operative Intubated, and Post-operative Extubated Arterial/Alveolar Oxygen Ratio

Mean			DF	SS	MS	F Ratio	
Pre-op	Post-op In	Post-op Ex					
.812	.526	.458	2	2.11	1.06	115.23	*S
			87	0.80	0.01		

*S=significance at alpha ≤ 0.05

Further one-way ANOVA subgroup analysis of smoking history, type of anaesthesia, albumin additive in pump prime, anaesthetic time, bypass time, cross-clamp time, right atrial pressure (RA), pulmonary capillary wedge pressure (PCWP), tidal volume, positive end-expiratory pressure (PEEP), cardiac index (CI), Dopamine, Nitroglycerin (NTG), Sodium Nitroprusside (SNP), time of suctioning, timing of the study, placement of chest tubes and left internal mammary artery graft (LIMA) on the shunt dependent variable revealed non significant findings for all these independent variables tested except for SNP and cardiac index. A significant difference was noted in shunt with subjects on SNP (M=13.45, F=4.38, DF=1,28). This indicates that SNP causes an increase in shunting and a decrease in PaO_2, regardless of positioning. Also significant differences were found in the cardiac index with shunt value (F-7.85, DF-2,27) and the mixed venous oxygen level (F-3.98, DF-2,27) in all three positions. Table 12 contains the statistical analyses of these subgroups.

Tests of the Hypotheses

Hypothesis I: Patients with left lower lobe atelectasis (LLL) confirmed by chest X-ray would have a lower arterial PO_2 and a higher relative shunt when placed in the left lateral position.

There were five subjects in which left lower lobe atelectasis was confirmed by chest X-ray. When these subjects were placed on their left side dependent, the PaO_2 was identical to the right lateral position and the relative shunt was lower than in the other two positions. The differences in each of these two variables among the three positions were not statistically significant (F-.015; p-.98; DF-2,12). This indicates that subjects with LLL atelectasis, when positioned on their left side, did not have their oxygenation altered. The data and the statistical analysis for these five subjects are listed in Table 13.

Hypothesis II: Patients with LLL atelectasis confirmed by chest X-ray would have a higher PaO_2 and a lower relative shunt in the supine and the right lateral positions.

There was no evidence to support this hypothesis. In these five subjects with LLL atelectasis, the PaO_2 value was higher in the right lateral position (M-133) than the supine position (M-128) but

Table 12

One-way Analysis of Variance of Subgroups

Smoking History and Relative Shunt (%)

Mean		DF	SS	MS	F Ratio	
Non-smoker	Smoker					
10.87	11.33	1	3.56	3.56	.26	N.S.
		28	386.05	13.79		

Type of Anaesthesia and Relative Shunt (%)

Mean		DF	SS	MS	F Ratio	
Analgesic	Analgesic & Inhalation					
9.31	11.78	1	35.00	35.00	2.39	N.S.
		28	396.88	14.17		

Albumin Additive in Pump Prime and Relative Shunt (%)

Mean Albumin		DF	SS	MS	F Ratio	
No	Yes					
10.93	11.82	1	8.08	8.08	0.53	N.S.
		28	423.81	15.14		

Sodium Nitroprusside (SNP) and Relative Shunt (%)

Mean SNP		DF	SS	MS	F Ratio	
No	Yes					
10.11	13.45	1	77.58	77.58	4.38	*S.
		28	496.15	17.72		

Nitroglycerin (NTG) and Relative Shunt (%)

Mean NTG		DF	SS	MS	F Ratio	
No	Yes					
10.10	11.59	1	9.17	9.17	0.45	N.S.
		28	564.56	20.16		

Dopamine and Relative Shunt (%)

Mean Dopamine		DF	SS	MS	F Ratio	
No	Yes					
10.89	15.56	1	47.94	47.94	3.50	N.S.
		28	383.94	13.71		

Left Internal Mammary Artery (LIMA) Graft and Relative Shunt (%)

Mean LIMA		DF	SS	MS	F Ratio	
No	Yes					
12.63	11.03	1	7.04	7.04	0.52	N.S.
		28	382.57	13.66		

Table 12 Con't

Positive End-Expiratory Pressure and Relative Shunt (%)

Mean PEEP (cmH_2O)			DF	SS	MS	F Ratio	
5.0	7.5	10.0					
11.05	10.79	12.76	2	17.92	8.96	0.65	N.S.
			27	372.03	13.78		

Anaesthetic Time and Relative Shunt (%)

Mean Anaesthetic Time (mins)		DF	SS	MS	F Ratio	
<300	>300					
11.65	10.60	1	16.12	16.12	1.21	N.S.
		28	373.49	13.34		

Right Atrial Pressure (RA) and Relative Shunt (%)

Mean RA Pressure (mmHg)		DF	SS	MS	F Ratio	
<12	>12					
12.14	10.65	1	24.75	24.75	1.90	N.S.
		28	364.83	13.03		

Tidal Volume and Relative Shunt (%)

Mean Tidal Volume (ml)			DF	SS	MS	F Ratio	
<1000	1001-1300	>1300					
8.93	10.55	12.19	2	35.43	17.72	1.35	N.S
			27	354.19	13.12		

Peak Airway Pressure (PAWP) and Relative Shunt (%)

Mean PAWP (cmH_2O)		DF	SS	MS	F Ratio	
<35	>35					
10.64	11.52	1	5.35	5.35	0.39	N.S.
		28	384.17	13.72		

Pulmonary Capillary Wedge Pressure and Relative Shunt (%)

Mean Wedge (mmHg)		DF	SS	MS	F Ratio	
<12	>12					
11.68	10.92	1	5.50	5.50	0.40	N.S.
		28	384.02	13.71		

Time Lapsed from Post-operation to the Study and Relative Shunt (%)

Mean Time (min)			DF	SS	MS	F Ratio	
<425	426-490	>490					
11.53	10.91	11.14	2	3.71	1.85	0.13	N.S.
			27	385.90	14.29		

Table 12 Con't

Time Suctioned Prior to the Study and Relative Shunt (%)

Mean Time (min)			DF	SS	MS	F Ratio	
<90	91-170	>170					
10.59	14.17	10.13	2	71.17	35.59	3.03	N.S.
			27	317.45	11.76		

Cardiac Index (CI) and Mixed Venous Partial Pressure Oxygen (mmHg)

Mean CI (L/min/BSA)			DF	SS	MS	F Ratio	
<2.3	2.31-3.0	>3.0					
34	35	38	2	84.74	42.70	3.98	*S.
			27	287.13	10.63		

Cardiac Index (CI) and Relative Shunt (%)

Mean CI (L/min/BSA)			DF	SS	MS	F Ratio	
<2.3	2.31-3.0	>3.0					
8.2	11.76	13.26	2	143.39	71.69	7.85	*S.
			27	246.44	9.13		

Bypass Time and Relative Shunt (%)

Mean Time (min)			DF	SS	MS	F Ratio	
<100	101-150	>150					
11.24	11.28	10.76	2	3.91	1.96	0.14	N.S.
			27	385.70	14.29		

Cross-clamp Time and Relative Shunt (%)

Mean Time (min)		DF	SS	MS	F Ratio	
<60	>60					
11.52	10.70	1	6.17	6.17	0.45	N.S.
		28	383.44	13.69		

*S-significance at alpha ≤ 0.05

Table 12 Con't

Location of Chest-tubes and Shunt

	Subject's Position		
Location	Supine	Right	Left
Left Pleural & Mediastinal	11.19	11.32	11.39
Right Pleural & Mediastinal	12.53	13.17	14.99
Mediastinal	8.44	7.69	8.71
Right, Left & Mediastinal	11.08	10.98	10.54
DF	3	3	3
	26	26	26
SS	13.13	24.89	56.64
	376.69	368.08	329.40
MS	4.38	8.30	18.88
	14.49	14.16	12.67
F Ratio	.30	.59	1.49
P*	.82	.63	.24

Location of Chest-Tubes and Arterial Oxygen

	Subject's Position		
Location	Supine	Right	Left
Left Pleural & Mediastinal	127	125	124
Right Pleural & Mediastinal	127	122	118
Mediastinal	146	153	145
Right, Left & Mediastinal	132	130	129
DF	3	3	3
	26	26	26
SS	435.53	934.10	697.76
	19247.17	22101.10	15097.60
MS	145.18	311.37	232.59
	740.28	850.04	580.68
F Ratio	.20	.37	.40
P	.90	.78	.75

*probability ≤ 0.05

equalled the PaO_2 value of the left lateral position ($M=133$). The shunt fraction was slightly lower in the right lateral position ($M=10.79$) than in the supine ($M=12.28$), and the left lateral shunt fraction ($M=10.59$) was the lowest value among the three positions. These differences were not statistically significant ($F=.20$, $DF=2,12$, $p=.824$).

Hypothesis III: Patients with no atelectatic changes on chest X-ray, would have no significant changes in PaO_2 and relative shunt in the three different positions.

The evidence to support this hypothesis was obtained from three subjects in the study who had a normal chest X-ray post-operatively. The PaO_2 ($F=.04$, $DF=2,6$, $p=.958$) and the shunt fraction ($F=.00$, $DF=2,6$, $p=.999$) showed no significant changes among the three positions Table 13.

Hypothesis IV: Patients with LLL atelectasis would have a higher relative shunt and a lower PaO_2 value when placed on the left lateral position due to an increase in cardiac output.

The arterial-venous oxygen content difference ($C[a\text{-}v]DO_2$) level did not differ significantly among the three positions ($F=.05$, $DF=2,12$, $p=.955$). The mean value of the $C[a\text{-}v]DO_2$ was slightly lower in the supine position ($M=4.97$) than the lateral positions ($M_R=5.19$, $M_L=5.04$) and this was reflected in the PaO_2 and shunt differences between supine and the two lateral positions. However, there is insufficient evidence to support this hypothesis in the study (Table 13).

Table 13

One-way Analysis of Variance of Chest X-ray Findings and the Dependent Variables

Left Lower Lobe Atelectasis and Arterial Partial Pressure Oxygen (mmHg)

Subject	Supine	Right	Left	DF	SS	MS	F Ratio	P*
2	93	92	101	2	80.13	40.03	0.07	.928
3	121	130	134	12	6422.81	535.23		
4	157	149	154					
10	144	144	147					
13	125	150	128					
Mean	128	133	133					

Left Lower Lobe Atelectasis and Relative Shunt (%)

Subject	Supine	Right	Left	DF	SS	MS	F Ratio	P
2	16.03	14.75	12.59	2	8.53	4.26	0.20	.824
3	13.92	9.73	10.00	12	260.87	21.74		
4	8.79	9.06	8.15					
10	5.07	5.52	4.92					
13	17.60	14.89	17.29					
Mean	12.28	10.79	10.59					

Left Lower Lobe Atelectasis and Arterial-Venous Oxygen Content Difference (ml O_2/100 ml)

Subject	Supine	Right	Left	DF	SS	MS	F Ratio	P
2	6.62	4.09	3.48	2	0.13	0.06	0.05	.955
3	4.45	5.06	4.78	12	16.86	1.40		
4	4.26	4.33	4.65					
10	6.75	6.45	6.91					
13	5.75	6.01	5.37					
Mean	4.97	5.19	5.04					

Normal Chest X-ray and Arterial Partial Pressure Oxygen (mmHg)

Subject	Supine	Right	Left	DF	SS	MS	F Ratio	P
7	144	153	156	2	60.63	30.31	0.04	.958
14	173	175	153	6	225.38	704.23		
15	117	117	117					
Mean	145	148	142					

Normal Chest X-Ray and Relative Shunt (%)

Subject	Supine	Right	Left	DF	SS	MS	F Ratio	P
7	6.28	5.76	5.79	2	0.05	0.02	0.00	.999
14	13.90	14.39	14.01	6	115.11	19.18		
15	12.32	12.64	13.22					
Mean	10.83	10.93	11.01					

Table 13 Con't

Normal Chest X-Ray and Arterial-Venous Oxygen Content Difference (ml O_2/100 ml)

Subject	Supine	Right	Left	DF	SS	MS	F Ratio	P
7	6.46	6.57	6.43	2	0.10	0.00	0.00	.998
14	4.94	4.73	5.34	6	7.80	1.30		
15	4.44	4.36	4.07					
Mean	5.28	5.22	5.25					

Right Lower Lobe Atelectasis and Arterial Partial Pressure Oxygen (mmHg)

Subject	Supine	Right	Left
19	93	93	90

Right Lower Lobe Atelectasis and Relative Shunt (%)

Subject	Supine	Right	Left
19	11.09	10.81	10.76

Right Lower Lobe Atelectasis and Arterial-Venous Oxygen Content Difference (ml O_2/100 ml)

Subject	Supine	Right	Left
19	6.18	6.37	6.52

Bilateral Atelectasis and Arterial Partial Pressure Oxygen (mmHg)

Mean			DF	SS	MS	F Ratio	P
Supine	Right	Left					
130	126	125	2	268.94	134.47	0.19	.823
			60	41381.31	689.69		

Bilateral Atelectasis and Relative Shunt (%)

Mean			DF	SS	MS	F Ratio	P
Supine	Right	Left					
10.97	11.36	11.40	2	4.31	2.16	0.16	.851
			60	797.41	13.29		

Bilateral Atelectasis and Aterial-Venous Oxygen Content Difference (ml O_2/100 ml)

Mean			DF	SS	MS	F Ratio	P
Supine	Right	Left					
5.12	4.97	4.78	2	1.23	0.62	1.08	.347
			60	34.26	0.57		

*probability ≤ 0.05

Hypothesis V: In patients with 25 % ECF excess, the different positioning would not change the PaO_2 and the shunt.

Twenty one subjects had 25% ECF excess, and these subjects had a lower shunt (M=10.25) and a higher PaO2 value (M=133 mmHg) than their counterparts. However, these differences were not statistically significant. Subjects with 25% fluid excess did not demonstrate any significant difference of the dependent variables among the three positions. This means that excessive fluid intake does not compromise oxygenation and that positioning in the presence of fluid excess is unimportant. Table 14 shows the analysis of 25% ECF excess and the dependent variables.

Correlations Among Relevant Variables

The data was subjected to further analysis with the Pearson's r correlation to determine the relationships among the different variables. The results showed some weak but significant correlation between some independent and dependent variables. The correlation matrix is illustrated in Table 15. Right atrial pressure had a positive correlation with pulmonary capillary wedge pressure (PCWP) and diastolic pulmonary artery pressure (PAP). Whereas, pulmonary capillary wedge pressure also had a positive correlation with systolic PAP. The pre-operative cardiac ejection fraction was found to have a weak negative association with the post-operative RA and wedge pressures. For those subjects with a good ejection fraction pre-operatively, the post-operative PAP pressures would likely be lower. This may imply that good contractility of the heart in the pre-operative period persists even after surgery. Anaesthetic

Table 14

One-way Analysis of Variance of 25% Extracellular Fluid Excess and the Dependent Variables

25% Extracellular Fluid Excess and Relative Shunt

Position	Mean <25%	Mean >25%	DF	SS	MS	F Ratio	P
Supine	13.00	10.39	1	42.70	42.70	3.44	.0740
			28	347.12	12.40		
Right	13.61	10.17	1	74.57	74.57	6.56	*.0161
			28	318.41	11.37		
Left	13.18	10.36	1	50.14	50.14	4.18	.0504
			28	335.90	12.00		

25% Extracellular Fluid Excess and Arterial Partial Pressure Oxygen

Position	Mean <25%	Mean >25%	DF	SS	MS	F Ratio	P
Supine	122	133	1	780.00	780.00	1.16	.2916
			28	18902.70	675.10		
Right	117	133	1	1803.74	1803.74	2.38	.1342
			28	21231.46	758.27		
Left	116	131	1	1429.53	1429.53	2.79	.1062
			28	14365.84	513.07		

*probability ≤ 0.05

Table 15

Correlation Matrix of Relevant Variables

Pulmonary Artery Pressures and Ejection Fraction

	PCWP1	PCWP2	PCWP3	PCWP4	PAD1	PAD2	PAD3	PAD4	EF*
RA1*	.6164 (29) .000**	---	---	---	.6198 (30) .000	---	---	---	-.3820 (26) .054
RA2	---	.5589 (29) .002	---	---	---	.7418 (30) .000	---	---	-.4922 (26) .010
RA3	---	---	.7020 (28) .000	---	---	---	.6653 (30) .000	---	-.4135 (26) .036
RA4	---	---	---	.7701 (29) .000	---	---	---	.7730 (30) .000	-.4558 (26) .019

	PAD1	PAD2	PAD3	PAD4	PAPSYS1	PAPSYS2	PAPSYS3	PAPSYS4	EF
PCWP1	.6899 (29) .000	---	---	---	.4213 (29) .023	---	---	---	-.4937 (26) .012
PCWP2	---	.6366 (29) .000	---	---	---	.4321 (29) .019	---	---	-.2835 (26) .170
PCWP3	---	---	.6808 (28) .000	---	---	---	.7420 (28) .000	---	-.3322 (26) .113
PCWP4	---	---	---	.6227 (29) .000	---	---	.5772 (29) .001	---	-.2046 (26) .327

*RA-Right atrial pressure; PCWP-Pulmonary capillary wedge pressure; PAD-Pulmonary artery diastolic pressure; PAPSYS1-Pulmonary artery systolic pressure; EF-Ejection fraction; Number 1, 2, 3, & 4 denotes the ordering of positions.

Subject's Characteristics and Inspiratory Oxygen with Shunt

	*QT1	QT2	QT3	QT4
Body surface area	.2777 (30) .137	.4352 (30) .016	.4683 (30) .009	.5270 (30) .003
Inspiratory oxygen	.5116 (30) .004	.5841 (30) .001	.5324 (30) .002	.5913 (30) .001

*QT-Shunt; Number 1, 2, 3, & 4 denotes the order of positions.

Table 15 Con't

Operative Time and Grafts

	Anaesthetic time	CPB* time	X-clamp time	Total grafts
Anaesthetic time	---	.7055 (30) .000	.7050 (30) .000	.5305 (30) .003
CPB time	---	---	.9704 (30) .000	.7236 (30) .000
X-Clamp time	---	---	---	.6920 (30) .000
Smoking years	---	---	---	.4325 (30) .024

*CPB-Cardiopulmonary bypass.

Subject's Characteristics

	Pack per Day	Albumin	Inspiratory Oxygen	Cardiac Output
AGE	-.4960 (28) .007	-.5252 (30) .003	---	-.5104 (30) .004
Smoking years	.6071 (26) .001	---	---	---
Body surface area	.3849 (28) .043	---	.3717 (30) .043	---
Weight change	---	---	---	-.4240 (30) .020

Positional Matched Tidal Volume and Peak Airway Pressure

	SPAWP	RPAWP	LPAWP*
STV*	.5960 (30) .001	---	---
RTV	---	.5299 (30) .003	---
LTV	---	---	.5283 (30) .003

*S-Supine position; R-Right lateral position; L-Left lateral position; PAWP-Peak airway pressure.

Table 15 Con't

Tidal Volume and Peak Airway Pressure

	PAWP1	PAWP2	PAWP3	PAWP4*
Tidal Volume	.4439 (30) .014	.4173 (30) .010	.4609 (30) .010	.3548 (30) .054

*PAWP-Peak airway pressure; Number 1, 2, 3, & 4 denotes the order of positions.

The Relationship Among Oxygen Content Indices and Oxygen Tension Index with Cardiac Output and Inspiratoy Oxygen

	a/AR1	a/AR2	a/AR3	a/AR4	CO	FiO_2*
QT1*	-.7646 (30) .000	---	---	---	.6276 (30) .000	.5116 (30) .004
QT2	---	-.8392 (30) .000	---	---	---	.5841 (30) .001
QT3	---	---	-.8647 (30) .000	---	---	.5324 (30) .002
QT4	---	---	---	-.8572 (30) .000	---	.5913 (30) .001
FiO_2	-.7099 (30) .000	-.7136 (30) .000	-.6892 (30) .000	-.7411 (30) .001	---	---
AVDO2	---	---	---	---	-.6099 (30) .000	---

*QT-Shunt; AVDO2-Arterial-venous oxygen content differences; CO-Cardiac output; a/AR-Arterial/Alveolar ratio; FiO_2-Inspiratory oxgyen; Number 1, 2, 3, & 4 denotes the order of positions.

Ejection Fraction with Fluid Balance and Recency of Myocardial Infarction

	Fluid Balance	Receny of MI*
Ejection Fraction	-.5400 (26) .004	-.2558 (26) .207

*MI-Myocardial infarction.

**probability $\leq$ 0.05

time, CPB, aortic cross-clamp time, and total grafts had positive relationships with each other. In other words, if the number of grafts increased, the anaesthetic time, CPB, and aortic x-clamp time also increased.

The number years of smoking was found to have some association ($r=.4325$, $p=.024$) with the number of grafts in the sample. Age was found to be inversely related to the number of cigarettes smoked daily ($r=-.4960$, $p=.007$), the post-operative albumin level ($r=-.5252$, $p=.003$), and the cardiac output ($r=-.5104$, $p=.004$). This suggests that with advancing age there was a tendency to smoke fewer cigarettes for this group of subjects. However, the significant negative correlation between age and the number of cigarettes smoked daily did not take into account those subjects who had ceased smoking many years ago. Therefore, the implied relationship between age and the number of cigarettes smoked daily is invalid. The correlation data also suggest that an increase in age is linked with lower post-operative cardiac functions and lower serum albumin levels.

Body surface area (BSA) was noted to be positively correlated with FiO_2 ($r=.3717$, $p=.043$) and relative shunt ($r=.4275$, $p=.041$). The size of the individual determines the oxygen requirement; large-sized individuals may have more areas of low $\dot{V}/\dot{Q}$ inequality, hence an increase in shunting. Additionally, FiO_2 was positively related to relative shunt ($r=.5548$, $p=.002$) and negatively correlated with a/A ratio ($r=-.7132$, $p<.001$), which means that an increase in oxygen concentration is required because there is more relative shunt. The relationship between relative shunt and arterial-alveolar ratio (a/A) was a negative correlation (r=-.8314, p<.001); as the relative shunt increased the a/A ratio decreased. On the other hand, a positive correlation was manifested between relative shunt and cardiac output (r=.6276, p<.001), which means shunting increases with a high cardiac output. An inverse relationship exists between cardiac output and arterial-venous oxygen content difference ($C(a\text{-}v)DO_2$) (r=-.6099, p<.001), signifying that an increase in the cardiac output results in a decrease in $C(a\text{-}v)DO_2$. The tidal volume was, as expected, positively correlated with peak airway pressure (r=.5514, p=.002).

CHAPTER V

Discussion of Findings

In this section, an overview of the findings related to the hypotheses and possible explanations for the findings are presented. The subgroup analyses are described, followed by the limitations of the study and implications of the findings for future nursing practice and research.

Summary of Major Findings

The major focus of the study was the effects of positioning on oxygenation. One-way analysis of variance (ANOVA) results showed no significant differences in the sequencing and treatment effects on the dependent variables. In other words, the order in which the patients were turned and the different positioning had no influence on oxygenation. The lack of significance of the sequencing effect has been supported by other researchers (Banasik et al., 1987; Shively, 1988). However, the non-significant treatment effect noted in this study was unexpected in view of previous studies in unilateral lung disease and in CABG patients (Zack et al., 1974; Dhainaut et al., 1980; Remolina et al., 1981; Ibanez et al., 1981; Hansan et al., 1982; Sonnenblick et al., 1983; Rivara et al., 1984; Banasik et al., 1987; Gillespie & Rehder, 1987). The findings of this study imply that positioning is not an important consideration for oxygenation. This conclusion is substantiated by studies using the mixed venous oxygen as an indicator of oxygenation (Shively, 1988; Banasik, 1990). In this study, there were no significant differences in the mixed venous oxygen saturation, the arterial oxygen saturation, and the arterial partial pressure carbon dioxide ($PaCO_2$) among the three positions further sustaining the conclusion. However, the treatment effect significantly altered some of the hemodynamic variables such as diastolic blood pressure, systolic pulmonary artery pressure, diastolic pulmonary artery pressure, and mean pulmonary artery pressure. The alteration in these variables was highest in both lateral positions with greatest increase in the left lateral position. The changes in these pressures might have been a significant component in the maintenance of stable relative shunt and arterial partial pressure oxygen (PaO_2) values in the three positions (Gillespie et al., 1990). The reasons for these particular pressure changes are obscure. The changes could be related to the reference point for the transducers, but no concurrent changes in systemic systolic blood pressure (BP), mean arterial BP, right atrial (RA) pressure, and wedge pressure were noted (Ross, 1989). Another possibility could be the migration of the pulmonary artery (PA) catheter in the lateral positions (Ross, 1989). Then, how would one explain the non-significant differences in RA and wedge pressures?

The overall non-significance in the treatment effect might be attributed to the different lung pathologies which existed among the sample. Subgroup analysis of the sample according to their lung pathology might be more meaningful. The first hypothesis was that when patients with left lower lobes (LLL) atelectasis were positioned on the left side, the PaO_2 and the relative shunt would be lowered. The second hypothesis was that when patients were placed on the right or in the supine position, the PaO_2 and the relative shunt would be higher. These two hypotheses were not supported by the data. In the subsequent subgroup analysis of the five subjects who had LLL atelectasis, three had a 3-9 mmHg PaO_2 escalation on the left side than in other positions. The other two subjects had an intermediate PaO_2 value among the three positions. Subjects two and ten both had a lower relative shunt and a higher PaO_2 value in the left lateral position. Subject four did not have a higher PaO_2 value in the left lateral position but had a lower shunt fraction. In the remaining two subjects, the shunt had a mid-range value among the positions. The mean change in shunting among the three positions was less than 3%. These findings completely contradict the expected results based on theory and previous research (West, 1977; Zack et al., 1974; Remolina et al., 1981; Sonnenblick et al., 1983; Banasik et al., 1987; Nunn, 1987; Dantzker, 1987). Comparing this sample with the sample of Banasik et al. (1987), one finds the attributes of the two samples are similar except in ventilation. Both groups of patients were electively ventilated after surgery, but in Banasik et al.'s study the patients were ventilated with a mean volume of 839 ml/breath, PEEP at 5 cm H_2O, and at a mean rate of 12/minute. In this study, all subjects were ventilated at a mean tidal volume of 1240 ml/breath, PEEP at 5 cm H_2O, and at a mean rate of 8/minute. This mode of high volume/low frequency with PEEP ventilation might have been the major reason for the lack of significant difference in the dependent variables among the three positions (Gillespie & Rehder, 1989).

Another hypothesis was that patients with LLL atelectasis, when placed in the left lateral position, would exhibit a lower arterial-venous oxygen content difference ($C(a\text{-}v)DO_2$) value which would result in a lower PaO_2 level and a higher shunt fraction. This hypothesis was based on the findings by Doering and Dracup (1988). They found a higher cardiac output in lateral positions, especially the left lateral position. Cardiac output holds an inverse relationship with $C(a\text{-}v)DO_2$. If a lower $C(a\text{-}v)DO_2$ occurred in the left lateral position, then an augmentation in perfusion of the left lung would be expected. The preferential location of atelectasis is the left lung bases in CABG patients; an increase in perfusion into the left lung would result in a decrease in the PaO_2 and an increase in the relative shunt. The findings contradicted the stated hypothesis. The data showed no consistent pattern that would suggest a higher cardiac output in the lateral positions or that an alliance existed among $C(a\text{-}v)DO_2$, PaO_2, and relative shunt in the left lateral position. The non-significance might again have been the overpowering effect of the high volume ventilation.

A further hypothesis was that different positions would not influence the dependent variables in patients with normal post-operative chest X-rays. The three subjects with normal chest X-rays supported the hypothesis, but the amount of shunting in these subjects was no different than the amount in those five subjects with LLL atelectasis.

Twenty-one subjects had bilateral atelectasis in the study; the relative shunt and the PaO_2 values were not significant in the three different positions. The supine position resulted in a lower mean relative shunt and a 6 mmHg higher PaO_2 value than the lateral positions. One subject had an X-ray abnormality in only the right lung, and this subject displayed a slight rise in the relative shunt and in PaO_2 value in the right lateral position.

The small sample size of subjects with normal and LLL atelectasis weakened the validity of the conclusion, especially when the effect size was small for the dependent variables. None of the changes in the dependent variables are statistically significant; nevertheless clinically these changes have a significance. The small improvement in these values might not be of importance in healthy patients. However, they are clinically important for post-CABG patients during the early period when the goal is to achieve an optimal oxygenation at a minimal level of supplemental oxygen to prevent any hypoxemic related complications and at the same time to prevent possible oxygen toxicity with a high FiO_2.

The persistent lack of statistical significance of the subgroup analyses in the presence of different lung pathologies might suggest that the mode of ventilation could be liable for the findings. Gillespie and Rehder (1989) point out that positional hypoxemia might not exist with a high tidal volume ventilation, but they offered no explanations. Little is known about the influences of high volume/low frequency ventilation with PEEP on pulmonary blood distribution and ventilation in the presence of lung pathology.

West (1977) provided some explanations for the observed findings in this study. Ventilation is dependent on the distensibility of the lung and the resistance of the airway. The differences in intrapleural pressure within the lung result in different resting lung volumes with a higher resting volume in the non-dependent areas and a lower volume in the dependent areas. The intrapleural pressure is dependent on the inspired volume. A high tidal volume ventilation increases the functional residual capacity (FRC) and the compliance of the alveoli and reduces the airway resistance. In other words, large tidal volume ventilation shifts the location of different lung regions on the pressure-volume curve to the right. This shift causes the upper lung regions to fall on the flat portion of the curve, resulting in a higher resting alveoli volume in the non-dependent area. The alveoli in these areas are near maximal inflation at rest, so the compliance of these upper alveoli is lowered. The lower lung regions are located on the steep portion of the pressure-volume curve which increases the compliance of these alveoli. In positive pressure ventilation a higher inflation pressure will be required to achieve the desired volume because of unequal areas of compliance and lung resistance. It follows that during inspiration, gas distribution would be directed primarily to the high compliance alveoli. In mechanical ventilation, the non-dependent lung is better ventilated than the dependent lung because of lesser opposing hydrostatic forces of the diaphragm (Nunn, 1987). In high tidal volume mechanical ventilation the gas distribution will be directed to the non-dependent lung, even though it is less compliance, before it is delivered to the dependent lung regions (Nunn, 1987; Robichaud, 1990; Krayer et al., 1989; Baehrendtz & Klingstedt, 1984). In essence, this suggests that a high volume ventilation over-inflates the non-dependent lung and optimally inflates the dependent lung (Bevan, 1990; Kahn et al., 1988).

The FRC is increased with a high volume ventilation and also with the PEEP (Nunn, 1987; Hubmayr et al., 1990; Good et al., 1979). In elderly patients, where airway closure occurs above FRC in an upright position, then the high volume ventilation prevents airway closure in the dependent lung regions by shifting the different lung regions to the right of the pressure-volume curve, thereby increasing the FRC (Nunn, 1987). Similarly, PEEP also prevents airway closure by the addition of a positive pressure at the end expiration to prevent the airways and alveoli from closing and increasing the FRC. In positive pressure ventilation at low tidal volume and low FRC, the airways and the alveoli in the dependent lung regions may close due to higher intrapleural pressure. The dependent lung regions, in this situation, are located at the lower end of the pressure-volume curve and the non-dependent lung regions are located at the steep portion of the curve. The compliance of the non-dependent lung is higher than the dependent lung. In high tidal volume positive pressure ventilation, there is an increase in FRC. The dependent lung regions in this situation are shifted to the steep portion of the pressure-volume curve and the non-dpendent lung regions are located on the upper flat portion of the curve. In other words, with high tidal volume ventilation the dependent lung has a higher compliance than the non-dependent lung, even though there is more resistance for diaphragm excursion. In this study, the functions of PEEP and of high volume ventilation appeared to be duplicated. This combination further decreases the compliance of the non-dependent regions and lowers the compliance of the dependent regions. The reason being is that with the addition of PEEP, the dependent lung regions are moving closer to the upper portion of the pressure-volume and the non-dependent lung regions are shifted to the flat portion of the curve. In addition, the increase in FRC can be augmented by elevating the head of the bed and by changing the patient to a lateral position (Nunn, 1987).

The high volume ventilation also alters the perfusion of the lung. Perfusion is influenced by gravity, but it is also dependent on the alveoli pressure and the compressive forces of the alveoli on the capillary. Normally, blood flow to the upper regions of the lungs is determined by the difference in alveolar pressure and pulmonary arterial pressure. In high tidal volume positive pressure ventilation (PPV), the alveoli in the non-dependent areas are over-inflated, this will raise the resistance of capillary blood flow, even though the extravascular vessel resistance is low. This is because the over-distended alveolar compresses and stretches the surrounding pulmonary capillary. Furthermore, in PPV the alveolar pressure is high at the non-dependent lung regions, thus the result is no perfusion through these capillaries, and forming the zone 1 condition described by West (1977). Blood shifted from these regions is directed to the dependent lung regions. In turn, it raises the pulmonary arterial pressure toward the dependent lung. The pulmonary artery pressure in the dependent lung exceeds the alveoli pressure. Perfusion through the capillary bed is determined by the difference in the pulmonary artery and venous pressures. In addition, the capillary resistance is less in the dependent regions because the alveoli are less distended. This mode of ventilation eliminated the low $\dot{V}/\dot{Q}$ regions and enhanced the matching of ventilation and perfusion--but it also increased dead space ventilation. The relatively stable shunt among the different positions in the study would suggest that the observed shunting was not the result of the low $\dot{V}/\dot{Q}$ regions but the zero $\dot{V}/\dot{Q}$ areas. It also showed that high tidal volume ventilation could not alter the zero $\dot{V}/\dot{Q}$ regions, suggesting that the collapsed regions in this group of patients might require a higher opening pressure to re-expand the atelectatic areas. The refractory shunt to oxygen therapy and hyperventilation indicates that shunting is associated with anatomical and capillary shunt (Shapiro et al., 1989).

Turning in bed may be seen as analogous to mild exercise for critically ill patients. Exercise causes a recruitment of collapsed capillaries and enhances matching of $\dot{V}/\dot{Q}$ in the upper regions of the lung (Ray et al., 1974; Tyler, 1984; Demarest et al., 1989; Gentilello et al., 1988). The frequent turning in this study might have prevented the occurrence of low $\dot{V}/\dot{Q}$ regions in the dependent areas and have improved the matching of $\dot{V}/\dot{Q}$ in the non-dependent area, explaining the lack of statistical significance observed among the positions. This confirms the findings of Ray et al. (1974), in which frequent turning was shown to have a protective effect in the pulmonary functions and improved the gas exchange.

The position of the patient will also influence perfusion and subsequent $\dot{V}/\dot{Q}$ inequalities. In an upright position, the vertical height of the blood column is great; pulmonary arterial pressure is lower in the upper lung regions (West,1977). In this study, the headrest elevation of 30° shortened the vertical distance of the blood column, which might have promoted a more even blood flow distribution throughout the lung with preference to the dependent lung regions. This would further minimize the $\dot{V}/\dot{Q}$ inequalities imposed by high volume ventilation with PEEP. Therefore, the high volume ventilation, combined with the elevation of the headrest and lateral positioning, might also have contributed to the stable shunt, because this combination increases FRC and minimizes dependent airway closure.

Another hypothesis was that in patients with 25% extracellular fluid (ECF) excess the PaO_2 and the relative shunt would not change with different positions. An increase in ECF volume would lead to an increase in filling pressure and cardiac output and a subsequent increase in perfusion to the lungs. The increased wedge pressure might promote extravascular fluid accumulation in the lungs in the presence of a low serum albumin level and a leaky capillary membrane (Weiland & Walker, 1986; Kowalski et al., 1989). The increased perfusion in the presence of the above condition might induce diffuse $\dot{V}/\dot{Q}$ inequalities in the lungs (Remolina et al., 1981). Positional hypoxemia would not exist in the presence of diffuse $\dot{V}/\dot{Q}$ inequalities (Ray et al., 1974). The data of 19 subjects who had 25% ECF excess did not demonstrate any significant change in the relative shunt or the PaO_2 values for the three positions. The other 11 subjects with less than 25% ECF excess did not show any significant positional fluctuations in the two dependent variables either. However, the 25% ECF excess group had a lower relative shunt and a higher PaO_2 than their counterpart. The likely explanation is the high tidal volume, since animal studies have shown that hypervolemia results in a lower shunt in the presence of a high volume ventilation (Smith et al., 1974).

Further support for the observed reduction in relative shunt comes from studies in which the alteration of flow and mixed venous blood results in changes in the amount of shunting (Smith et al., 1974; Bishop & Cheney, 1983; Cheney & Colley, 1980; Kelmen et al., 1967; Philbin et al., 1970). An excess of ECF would increase the preload and subsequently increase the cardiac output. An increase in cardiac output would increase the pulmonary arterial pressure and the mixed venous oxygen level when oxygen consumption is constant. The increase in cardiac output and in mixed venous blood oxygen would increase perfusion to all regions of the lungs and inhibit the hypoxic pulmonary vasoconstriction (HPV) of the low $\dot{V}/\dot{Q}$ regions, resulting in an increase in shunting. In the presence of high volume/low frequency ventilation, the increase in ECF volume and preload would result in an increase in pulmonary artery pressure. The increase in the pulmonary arterial pressure would minimize the dead space ventilation by recruiting collapsed capillaries caused by the over-distended alveoli and would enhance a more even blood distribution. The improvement in $\dot{V}/\dot{Q}$ matching would compensate for the other adverse effects of increased cardiac output. On the other hand, a lower ECF excess volume would mean that the pulmonary artery pressure would be lower and result in a disproportionate flow in the dependent regions where the zero $\dot{V}/\dot{Q}$ areas are localized, thus explaining the higher relative shunt and lower PaO_2 values.

Additional Findings

The demographic data of this group of subjects were similar to the data of Banaski et al.'s (1987) and Shively's (1988) studies. In this study, overweight male subjects predominated; a history of smoking and angina pectoris were prevalent. The mean ejection fraction was less than normal in this sample but at a range that posed no extra risk to perioperative mortality rate (Rutherford & Braunwald, 1988). The mean pulmonary function tests were normal in this sample. Pearson's r correlations were done with some of the demographic data, and some statistically significant correlations were found.

Age was found to have a weak negative correlation with postoperative cardiac output. This finding was expected, because the aging process causes a decrease in the elasticity of the vessels, a dilation of the venous system secondary to muscle atrophy, and a decrease in contractile force of the cardiac muscle (Underhill et al., 1984). All these lead to an elevation in systematic vascular resistance and a reduction in venous return to the heart (Weeks, 1986). Furthermore, the subnormal ejection fraction of this group of patients, combined with the insult of the surgery (Boldt et al., 1987), explained the observed inverse relationship between age and cardiac output. Another negative correlation existed between age and the post-operative serum albumin level. The low normal serum albumin level may suggest a deficit in protein intake of the aging population. Hemodilution might also reduce the serum albumin level (Kowalski et al., 1989; Rein et al., 1988), but the serum osmolality was within normal limits. The number of packs per day was positively correlated with smoking years; this might be the tolerance of the nicotine level in the body. In other words, the consumption of cigarettes increases with the duration of smoking years. An interesting finding was a weak significance between smoking years and the number of coronary bypass grafts. The possible explanation is that cigarette smoking is associated with an increased risk of atherosclerosis of the aorta and the coronary arteries (Sybers, 1986).

The physical size of an individual was considered an influential factor. The body surface area (BSA) was confirmed to have a positive correlation with the FiO_2, indicating that the size of an individual determined the oxygen requirement. The subjects in this study were sedated, and the mixed venous oxygen did not change significantly throughout the study, implying that in large body size individuals there may be more regions of lung pathology. Lung pathology creates $\dot{V}/\dot{Q}$ inequalities. The existence of $\dot{V}/\dot{Q}$ mismatches is the major cause of higher oxygen requirement in subjects with a high BSA. The BSA was also found to be positively correlated with the relative shunt fraction. The BSA value is calculated based on the height and weight values. An increase in the body weight increases the BSA value. In the study, the overweight subjects carried most of their excess weight around the abdominal area. The distended abdomen increases the resistance for diaphragmatic excursion, hence reducing the FRC in the dependent regions and promoting atelectasis formation (Nunn, 1987; Ericksen, 1978; Jenkins et al., 1988).

The pre-operative ejection fraction was negatively correlated with the post-operative fluid balance. The ejection fraction represents the cardiac output; this fraction would be reduced after surgery because of the ischemia connected with cross clamping and the myocardial depression with topical cooling (Curtis et al., 1989). The kidney receives 20% of the cardiac output. Reduction in cardiac output would reduce the ability of the kidney to get rid of the excess fluid, explaining the inverse relationship between these two variables. Surprisingly, there was no correlation between the recency of myocardial infarction and the ejection fraction. The ejection fraction was also negatively correlated with the right atrial (RA) pressure and the wedge pressure, indicating that with left ventricular failure, a forward pumping failure, there is a backward build up of pressure in the system (Underhill et al., 1982). The RA, wedge, and diastolic pulmonary artery (PAPD) pressures were positively correlated with each other, while wedge pressure was also positively correlated with systolic pulmonary artery pressure (PAPS). There is an inter-relationship among these pressures. They are in serial to each other, hence when one pressure rises the others will follow. The cardiac output is negatively correlated with $C(a\text{-}v)DO_2$. The reasons for this inverse relationship have been previously discussed. The cardiac output is also found to be positively correlated with the relative shunt fraction. This connection would suggest that an increase in cardiac output increases perfusion to all lung regions with preferential perfusion to the dependent lung where low $\dot{V}/\dot{Q}$ and atelectatic regions are located. Furthermore, an increase in cardiac output will increase mixed venous blood oxygen. This increase in PvO_2 will inhibit the HPV response resulting in venous admixture (Cheney & Colley, 1980; Kelman et al., 1967; Philbin et al., 1970; Smith et al., 1974; Bishop & Cheney, 1983). The shunt had a strong inverse relationship with the a/A ratio; the FiO_2 had a positive correlation with the shunt fraction and an inverse relationship with a/A ratio. The inter-relationship among shunt, a/A ratio, and FiO_2 confirmed the findings of previous studies (Douglas et al., 1979; Shoemaker et al., 1989).

The results of the pre-operative pulmonary function tests had no connection with any post-operative functions or the duration of post-operative intubation. The value of routine pulmonary function testing in patients without history of lung abnormality needs to be re-evaluated. However, the ventilated tidal volume has a positive correlation with the peak airway pressure (PAWP), meaning that a higher pressure is required to deliver a larger volume because the compliance of the lung declines after an optimal lung volume has been achieved (Nunn, 1987).

Atelectasis is unrefutedly common among post-operative CABG patients. The incidence of immediate post-operative atelectasis in this study was 90%, with bilateral atelectasis being most common.

This result differs from other studies (Banasik et al., 1989, Jansen & McFadden, 1984; Jenkins et al., 1988) in which LLL atelectasis is the most common post-operative pulmonary complication for CABG patients. The etiology of the differences might be due to the types of anaesthetic, the cardiopulmonary bypass (CPB) machine, and the operative techniques. Nevertheless, the incidence of 27 subjects with x-ray changes in the immediate post-operative period was a cause for concern. The incidence of atelectasis did not change on post-operative day one, indicating that the mode of ventilation in this group of subjects did not resolve the collapsed area. It further signified that there might have been persistent effects of the anaesthetic, CPB, and the surgery in the post-operative period. The deterioration found by X-ray on post-operative day two and day three implied that the high volume/low frequency ventilation did not resolve the atelectatic area but prevented its progression. Once post-CABG patients were extubated, the extraneous factors of incisional pain, altered muscle integrity, and the stability of the chest wall may have contributed to an increase in $\dot{V}/\dot{Q}$ inequalities and subsequent atelectasis (Jansen & McFadden, 1986; Jenkins et al., 1989). The significant results of a/A ratio among pre-CABG, intubated post-CABG, and extubated post-CABG periods provide evidence to demonstrate the efficiency of high volume ventilation in preventing atelectasis progression and the adverse effect of this mode of ventilation (Lee et al., 1990; Haake et al., 1987; Kolobow et al., 1987; Greenfield et al., 1964; Glauser et al., 1988).

The small sample size precludes any show of statistical significance in some of the variables that might have shown some significance in a larger sample. Nevertheless, some of these variables will be described because of their subtle differences.

There were two types of anaesthetic used in this sample. One group of seven subjects received an analgesic anaesthesia and the other group received a combination of inhalation and analgesic anaesthesia. The analgesic anaesthetic group had a lower relative shunt value than the combined group. The likely explanation is that inhalation anaesthetics decrease FRC and blunt the HPV responses (Hedenstierna et al., 1986; Schwieger et al., 1989; Baehrendtz & Klingstedt, 1984). It is unclear as to the duration of these effects, but from this study it appears that they were still present at 12 hour post-anaesthesia.

Albumin as the CPB pump additive was changed in the middle of the study, so that nine subjects received and 21 subjects did not receive albumin in their pump fluid. An interesting observation was noted: those who did not receive albumin had a lower relative shunt fraction. The probable reason for this is that during CPB, there is activation of the complement system which results in altered capillary permeability (Matthay & Wiener-Kronish, 1989; Weiland & Walker, 1986). At the completion of CPB, reperfusion of the lung in the presence of extra serum albumin may lead to leakage of albumin through the damaged membrane into the alveoli, especially in the dependent regions, thereby increases in the synthesis of zero V/Q areas were noted (Weiland & Walker, 1986).

The common inotropic and vasoactive drugs have been shown to increase shunting. The findings in this study support the idea that vasoactive agents increase shunting (Cheney & Colley, 1980; Radermacher et al., 1989). There were 11 subjects on Sodium Nitroprusside (SNP) and the amount of relative shunting was significantly higher than for those who were not on SNP. The increase in relative shunting by SNP is primarily because of the inhibition of the HPV response. Similar results were obtained with NTG, but the difference in shunt value was non-significant between those subjects who were on NTG and those who were not on NTG. NTG have been demonstrated to have a similar action to SNP, but it is not as potent (Nomoto & Kawamura, 1989; Radermacher et al., 1989).

Dopamine is the most commonly used drug in CABG patients. In this study, only two subjects did not receive this drug. Dopamine has been shown to increase cardiac output and to increase mixed venous oxygen with the repercussion of an increase in shunting and worsened oxygenation (Rennotte et al., 1989; Shoemaker et al., 1989; Nomoto & Kawamura, 1989). However, the opposite findings were obtained in this study. The two subjects who were not on Dopamine had a higher relative shunt than those who were on this drug. However, these two subjects were on SNP and NTG, which might explain the high relative shunt value.

The use of the left internal mammary artery (LIMA) and the entry into the pleural space are thought to increase shunting (Jenkins et al, 1989). In this study, the location of the chest tubes appeared to make a slight difference in the two dependent variables. One subject had mediastinal chest-tubes; this subject had a relative shunt of less than 10% and a high PaO_2 value. Subjects whose pleural space had been entered had a shunt of greater than 11% but the PaO_2 value did not appear to be affected. Twenty-seven subjects had LIMA graft as a conduit, and only three had exclusive saphenous vein grafts. A lower shunt was observed in patients with LIMA grafts. No possible explanations can be given for this observation.

When the cardiac index was tested with the relative shunt and the mixed venous oxygen level, significant correlations were noted. This supports the findings in animal studies where increases in flow results in increases in shunting because of the augmented perfusion to the atelectatic area and the inhibition of HPV response by the elevated PvO_2 (Bishop & Cheney, 1983; Smith et al., 1974; Cheney & Colley, 1980; Kelman et al., 1970).

The shunt value is dependent on the right atrial (RA) pressure, the wedge pressure, and the ECF volume excess. When RA and wedge pressures were greater than the normal value of 12 mmHg the relative shunt decreased. A similar finding was obtained with ECF excess, as discussed before. Higher filling pressures are needed to compensate for the adverse effects of positive pressure ventilation (PPV) with a high volume and positive end-expiratory pressure (PEEP) (Smith et al., 1974). The effects of peak airway pressure (PAWP), tidal volume, and PEEP on the dependent variables were analyzed. Shunting increased with an increase in PAWP, tidal volume, and PEEP (Lee et al., 1990). This shows that a high volume ventilation with PEEP does have an adverse effect on gas exchange, but the adverse effects are minimized by the other factors discussed above.

A history of smoking is prevalent among CABG patients, and this variable was tested with the relative shunt variable. Non-smokers had a lower shunt than their counterparts, although the difference was not significant. Smoking is believed to increase the closing volume and blunt the HPV response (Seaton, 1979).

In conclusion, positional hypoxemia does not occur in CABG patients who are ventilated with high tidal volume/low frequency and PEEP. It appears that this mode of ventilation improves the $\dot{V}/\dot{Q}$ ratio and prevents progression of atelectasis formation during the first 24 hours of high volume/low frequency ventilation. There is a constraint to this statement in that it is only true when the patients are hypervolemic with good cardiac output. The use of vasoactive drugs will alter oxygenation even in the presence of this mode of ventilation.

Limitations of the Study

The conclusions of this study are limited by several aspects of the study design. The convenience sample of CABG patients precludes the generalization of the findings to other patient populations. The exclusion criteria reduces the external validity even to the same patient population. The findings are exclusive to CABG patients with similar characteristics, and this must include the mode of ventilation.

The turning frequency in this study is not a standard of practice in any unit. Again the findings are limited to CABG patients who are being turned at the same frequency. The generalizations of the finding can not be extended to CABG patients who are being turned every two hours. The rapid turning in this study might have been a deterrent for a low $\dot{V}/\dot{Q}$ region to form. Findings might have been completely different if the subjects had been turned every two hours. Another shortcoming of this study rests with the initiation of the study. Most of the subjects were studied within 12 hours post-operation. Post-operative lung abnormalities peak at 24 hours. X-ray changes were found to be stable on day zero and day one, the reason for this being that manifestation of X-ray changes has a 24 hour lag time unless the change is dramatic. The study might have underestimated the severity of relative shunting and the value of positioning.

The 30° head of bed elevation was established at the start of the study and the transducers were levelled to the phlebostatic axis. Some patients tended to migrate to the bottom of the bed, and this might have changed the accuracy of the 30° headrest elevation and reference level for the transducers. The precision of the 30° trunk rotation was difficult to achieve, as some patients shifted their positions either higher or lower than the prescribed angle. This shift might limit the reliability of the study.

In addition, the X-ray changes of the subjects were based on the official radiologists' reports. These radiologists had no information that a study was being conducted. Consequently, the limitation was the lack of documentation of the relative changes between the lungs. Furthermore, the inconsistent use of terminology in reporting made it difficult at times to extrapolate information from the report.

Lastly, the small sample size of this study, especially in groups of subjects with LLL atelectasis, RLL atelectasis, and no X-ray changes, limit the external validity of the study.

Implications for Practice

Positioning in the presence of a high volume/low frequency and PEEP ventilation is not an important consideration, even with lung pathology. However, frequent turning may prevent the formation of low $\dot{V}/\dot{Q}$ regions in this group of patients. The progressive deterioration in X-ray findings and a/A ratios after extubation might suggest that frequent turning in this period is more important than during the intubated period.

The use of PaO_2 to determine the adequacy of gas exchange may be misleading. Relative shunt calculated at clinically useful FIO_2 levels may provide more relevant information as to the extent of the gas exchange abnormalities. In the absence of a pulmonary artery catheter, the a/A ratio may be used to monitor the gas exchange deficit. In caring for CABG patients who are ventilated with this mode of high volume/low frequency with positive end expiratory pressure ventilation, changes in the relative shunt and arterial oxygen level may be attributed to factors such as hydration status, cardiac output, vasoactive agents, and oxygen consumption. A systematic evaluation of these variables may assist in the identification of possible causes for the change in arterial oxygenation. It is important to assess the total patient so that the etiology of hypoxemia is dealt with rather than just the treatment of low arterial partial pressure oxygen.

The body surface area of individuals is also an important consideration in arterial oxygenation. Obese individuals are more prone to have lung pathology because of a lower functional residual capacity secondary to an increased resistance for diaphragmatic excursion. Consequently, in obese patients, lateral positioning increases diaphragmatic excursion which may improve arterial oxygenation. Hence, based on this study, factors that nurses need to consider in the event of changes in arterial oxygen and relative pulmonary shunt are fluid status, cardiac output, vasoactive drugs, obesity and frequency of turning.

Lastly, the supine phlebostatic axis as the levelling point for the transducers in lateral positions has proved to be a reasonable reference point for monitoring the hemodynamic variables. An augmentation of the cardiac output in lateral positions has not been demonstrated. However, measurement of cardiac output should be carried out in the supine position for consistency in the evaluation.

Implications for Future Research

Replication of this research using a different turning frequency would be necessary if the generalization that positioning is not an important consideration in the presence of high volume/low frequency and PEEP ventilation is to be accepted. Timing a study at 24 hours post-CABG might provide more insight as to whether positioning is important, because lung abnormalities peak at this interval. Furthermore, a follow up study, whether at 24 or 48 hours post-extubation, might provide additional information as to the after effect of this mode of ventilation on gas exchange. Since this mode of ventilation is not commonly used, there should be a retrospective chart review to determine its efficiency in the incidence of lung complications. In the institution where the study was conducted, it is common knowledge that CABG patients who require ventilation for more than the normal 24 hours have increased incidence of lung infection. Thus, a prospective study of the lung complications of CABG patients who are ventilated in this mode for longer than three days would provide information as to the adverse effects of this type of ventilation. In addition, the use of positioning and this mode of ventilation as a therapeutic means for re-expansion of the collapsed lung regions would be a useful study for this group of patients with a high incidence of atelectasis.

Lastly, the effect size of the dependent variables is small. A larger sample size of CABG patients and in different patient populations would strengthen the internal and external validity of the conclusion.

Conclusion

The purpose of this study was to investigate the effects of three different positions on arterial oxygen and relative pulmonary shunt in ventilated CABG patients. A repeated measures design was conducted in a convenience sample of 30 CABG patients. The study protocol was initiated within 12 hours post-operation. The findings of the immediate post-operative chest X-ray showed 90% of the subjects had atelectasis. However, the data revealed non-significant results in sequencing and positioning effects on arterial partial pressure oxygen and relative shunt. The lack of significant findings with the positioning effect was unexpected in view of the theory of pulmonary physiology and previous studies of unilateral lung diseases. The likely explanation for the non significance in this study is the mode of ventilation. This group of subjects was ventilated on a high volume/low frequency and positive end-expiratory pressure. This mode of ventilation might have prevented the formation of low ventilation/perfusion regions and delayed the progression of atelectasis formation. There is evidence to suggest that this mode of ventilation in the presence of hypervolemia, adequate cardiac output, frequent positioning, and slight headrest elevation may improve the ventilation/perfusion ratio. The stable relative shunt value and arterial oxygen level among the three positions found in this study may imply that positional hypoxemia does not exist in CABG patients who are ventilated with this mode of ventilation.

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APPENDIX A

Extracellular Fluid Volume Calculation

	Male	Female
Total Body Water (percent of body weight)	60%	50%
Compartment		
Intracellular Volume	2/3	2/3
Extracellular Volume	1/3	1/3

Example:

Pre-operative Weight of a male patient 70 kg

Total Body Water: 60% x 70 kg = 42 kg (1 kg = 1 L)
therefore 42 L

Intracellular Volume: 2/3 x 42 L = 28 L

Extracellular Volume: 1/3 x 42 L = 14 L

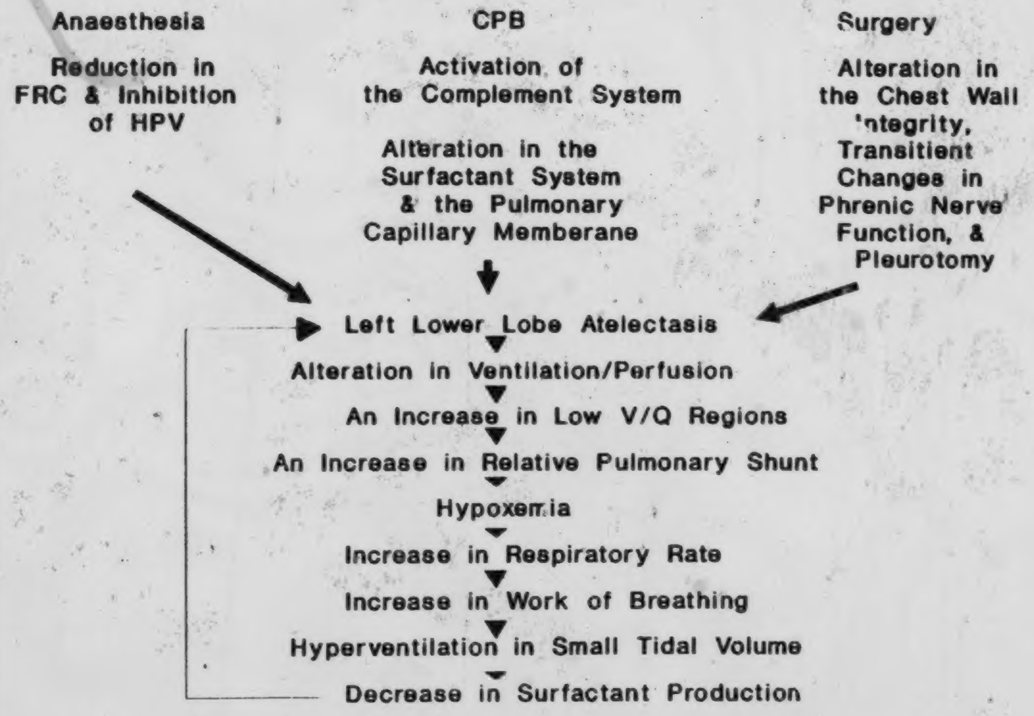
Post-operative Fluid Balance: + 5 L

Extracellular Volume Excess:

$$\frac{(14\ \text{L} + 5\ \text{L}) - 14}{14} \times 100\% = 35.71\%$$

APPENDIX B

Conceptual Framework of the Study
The Pathogensis of Atelectasis in CABG


Anaesthesia
Reduction in FRC & Inhibition of HPV
CPB
Activation of the Complement System
Alteration in the Surfactant System & the Pulmonary Capillary Memberane
Surgery
Alteration in the Chest Wall Integrity, Transitient Changes in Phrenic Nerve Function, & Pleurotomy
Left Lower Lobe Atelectasis
Alteration in Ventilation/Perfusion
An Increase in Low V/Q Regions
An Increase in Relative Pulmonary Shunt
Hypoxemia
Increase in Respiratory Rate
Increase in Work of Breathing
Hyperventilation in Small Tidal Volume
Decrease in Surfactant Production

APPENDIX C

Sample Selection Criteria

The pre-operative selection criteria:

1. **Adults Between age 50-70 years**: Aging affects pulmonary functions. This range is utilized to control for alteration in oxygenation secondary to normal physiological changes.
2. **Elective CABG surgery**: Emergency CABG surgery patients will not be approached because they may be hemodynamically unstable in the post-operative period. In an emergency situation there may not be an opportunity for an informed consent, therefore only elective CABG patients will be included.
3. **Number of coronary bypass grafts between one to five**: The number of bypass grafts is correlated with the length of cardiopulmonary bypass (CPB) time. Researches have shown that long CPB time may influence oxygenation (Weiland & Walker, 1986; Wilcox et al., 1988).
4. **English speaking**: The patient must understand English in order for the researcher to explain the study to him or her.
5. **Capable of signing an informed consent**: The capability of the patient will be determined by his or her orientation to time, place and person. The consent for the CABG surgery must be signed by the patient.
6. **Absence of documented pulmonary disease in the pre -operative period**: Existing pulmonary disease may cause

changes in pulmonary physiology and therefore may affect the observation on oxygenation in the post-operative period.

7. **Absence of myocardial infarction in the past six months**: Myocardial infarction depresses the contractility of the heart. There is evidence to show that low contractility persists up to six months after an infarct (Underhill et al., 1982). This may cause heart failure which will lead to pulmonary congestion and, in turn, may alter the relative pulmonary shunt and oxygenation.

8. **Absence of valvular disease or previous valvular replacement**: Valvular diseases will change the physiology of the cardiac and the respiratory system.

The post-operative selection criteria

1. **Hemodynamically stable with systolic blood pressure greater than 90 mmHg**: The subject has to be hemodynamically stable in order to have adequate perfusion to all organs. Hypotension (less than 90 mmHg systolic) will affect the SvO_2 results.

2. **Mechanically ventilated**: Mechanical ventilation will allow accurate control of the oxygen concentration and facilitate monitoring of the inspiration pressure of the lung. The subject will follow the protocol of the unit for respiratory management. The ventilator settings will be set at: synchronized intermittent mandatory ventilation (SIMV), rate between eight to fourteen per minute, tidal volume of 15 ml/kg of pre-operative body weight, positive end-expiratory

pressure (PEEP) of five centimetre of water and fractional inspired oxygen (FiO_2) will be individually determined.

3. Absence of external cardiac assist device such as intra-aortic balloon pump: Use of external cardiac assist devices will prevent the subject turning from side to side.
4. Body temperature greater than 36.5°C and 38°C: Pyrexia will increase oxygen consumption and in turn will affect the relative shunt calculation.
5. Presence of an arterial line and a pulmonary artery catheter: These two lines must be present and functioning properly in order to carry out the study.

Revisions to Sample Selection Criteria

The pre-operative selection criteria:

1. Adults scheduled for CABG surgery
2. Elective CABG surgery
3. Number of coronary bypass grafts between one to five
4. English speaking
5. Capable of signing an informed consent
6. Absence of pulmonary disease in the pre-operative period
7. Absence of myocardial infarction in the past six weeks: Myocardial infarction depresses the contractility of the heart. There is evidence to show that low contractility persists up to six weeks after an infarct (Underhill et al., 1982). This may cause heart failure which will lead to pulmonary congestion and, in turn, may alter the relative pulmonary shunt and

oxygenation. Recent data show that the interval between myocardial infarction and CABG surgery is not an important factor in stable patients less than 60 years old (Kennedy et al., 1989).

8. Absence of valvular disease or previous valvular replacement

The post-operative selection criteria

1. Hemodynamically stable with systolic blood pressure greater than 90 mmHg.
2. Mechanically ventilated
3. Absence of external cardiac assist device such as intra-aortic balloon pump
4. Body temperature greater than 36.5°C
5. Presence of an arterial line and a pulmonary artery catheter

APPENDIX D

Informed Consent

The University of Alberta
Faculty of Nursing

Topic of the Study

The Effects of Three Different Positions
on Arterial Oxygen and Relative Pulmonary Shunt
in Post-Operative Coronary Artery Bypass Graft
Patients

Purpose of the study:

Turning patients after surgery is done to promote comfort and to loosen secretions. After coronary artery bypass surgery, it is unclear if there is a certain position or positions that may improve blood oxygen. In this study the effects of three different positions on blood oxygen will be studied. Taking part in this study may help other patients after this surgery to find positions that will improve blood oxygen.

Procedures:

After your surgery, you will be in the Cardiovascular Intensive Care Unit. This study will take place in this unit on the day of your surgery. The study will start six to ten hours after the surgery. During the time of the study you will have a breathing tube in your mouth connected to the breathing machine (ventilator). You will also have a line in your neck (a pulmonary artery catheter) and another one in your wrist (an arterial line). These are put in during surgery. First the researcher will take a small amount of blood, in the usual manner, from the line in your neck and in your wrist. This will not hurt. After the blood is take, you will be helped to turn by the researcher and another person. You will be turned two more times in the study. After 30 minutes in each new position a small amount of blood will be taken. Taking blood from these lines is done on all patients after this surgery. The researcher is a nurse who is able to take blood from these lines.

You will be turned three times, 30 minutes apart in the study. The three different positions are: the left side, the right side and the back. Turning you every 30 minutes is more than what is usual after this surgery. There will be pain when you are turned. You will be give a painkiller before you are turned and at the end of the study. This will help to decrease the pain when you are turned.

Voluntary Participation:

Taking part in this study is up to you. If you decide to be in the study and change your mind later, you can drop out of the study at any time by raising your hand to the researcher. Your nurses in the intensive care unit can also ask the researcher to stop the study if they feel you are in too much pain. Your care during your hospital stay will not be affected if you are or are not in the study.

Confidentiality:

Your name and any information obtained from your chart will be kept private. Records fro this study will not be marked by your name but with a code number. Your name and code number will be kept locked in a cupboard. After the study is over, your name and code number will be destroyed. Your name will not be included in any reports of this study, nor in any articles or talks about the study.

If your have any questions, the researcher will be happy to answer them now. If you have questions later, you can contact the researcher or the researcher's supervisor at the Faculty of Nursing by phone.

Participant's Statement:

I, ____________________________, have read this information and agree to be in this study. I give permission to the researcher to look at my hospital record. I have had the chance to ask questions about the study and my part in it. The researcher, Monica Chan, has answered all my questions at this time. I have also been give a copy of this consent form.

Signatures:

Subject ____________________ Researcher ____________________

Date ________________________

APPENDIX E

Data Collection Sheet

Patient Data:

Patient Code Number: ..... Date of Admission: ....
Date of Surgery: .... Diagnosis: ....
Sex: .... Age: ....
Height: .... Weight: ....
BSA: .... Hgb: ....

Relevant Medical History: (angina, diabetes, hypertension etc.)
....
....
Smoker (years): .... Pack per day: ....
Quit (mos): ....

Preoperative ABG:
....

Pulmonary function tests:

FEV1 .... FVC .... FEV1/FVC ....
EF ....

Operation Events:

	Start	Finish	Total
Anaesthetic time:	....	....	....
Bypass time:	....	....	....
X-Clamp time:	....	....	....
Chest tubes	....		

Fluids:

Crystalloid .... Colloid ....
Cardioplegia .... Pump prime ....
Fluids given by perfusionist ....
Albumin .... Mannitol ....
Autotransfusion .... Total ....

EBL .... Urine output: ....
Insensible loss .... Third space fluid ....
Total output: .... Fluid balance: ....

Pump temperature in OR:
Lowest .... Highest ....

Complications in the OR
....
.... The Study Day:

Time of study: Start........ Finish ....
Total ....

Number of hours post-op: ....

Number of grafts: ...... SVG: .... IMA: ....

Post-op weight: .... Change in weight: ....

Blood work: Hgb .... Hct .... HCO2 ....
Osmo .... Ablumin ....

Vasoactive Medications
(Dosage)..
Diuretics: .... Time given: ....
Analgesic: .... Time given: ....

Type of IV fluids: ....
Total IV fluids per hour.......
Autotransfusion ....
IV fluids post-op .... Colloids post-op ....
Chest tube losses .... Urine output ....
NG .... Hemovac ....

Total crystalloid .... Total Colloid ....
Total Input ....

Total chest tube losses ...... Total urine output ....
Total NG losses .... Total Hemovac ....
Total Output .... Balance ....

Respiratory:

Ventilator setting:

Mode: .... Rate: .... FiO2 ..... PEEP ....
Tidal Volume: ....
Time of last suctioning: ....
Amount of Secretion: ....

Environmental Conditions:

Room Temperature: ......
Activity level: ..... Noise level:....

Initial Position: ....
Duration in the initial position: ....
Position Sequencing Schedule: ....

Position ....

Time (mins)	0	Event	5	Event	15	Event	30	Event
Time								
Hemodynamics:								
HR	.....		.....		.....		.....	
BP	.....		.....		.....		.....	
MAP	.....		.....		.....		.....	
PAP	.....		.....		.....		.....	
MPAP	.....		.....		.....		.....	
PCWP							.....	
RA	.....		.....		.....		.....	
CO	....							
Temperature	....							
PVR	....			SVR				
Respiratory:								
Rate	.....		.....		.....		.....	
VT	.....		.....		.....		.....	
PAWP	.....		.....		.....		.....	
Minute Vent	.....		.....		.....		.....	

Position ....

Time (mins)	0	Event	5	Event	15	Event	30	Event
Time:								
HR	.....		.....		......		.....	
BP	.....		.....		......		.....	
MAP	.....		.....		......		.....	
PAP	.....		.....		......		.....	
MPAP	.....		.....		......		.....	
PCWP							.....	
RA	.....		.....		......		.....	
Respiratory:								
Rate	.....		.....		......		.....	
VT	.....		.....		......		.....	
PAWP	.....		.....		......		.....	
Minute Vent	.....		.....		......		.....	

Position ....

Time (mins)	0 Event	5 Event	15 Event	30 Event
Time:				
HR	.....	.....	......	.....
BP	.....	.....	......	.....
MAP	.....	......	......	.....
PAP	.....	......	......	.....
MPAP	.....	......	......	.....
PCWP				.....
RA	.....	......	......	.....
Respiratory:				
Rate	.....	......	.....	......
VT	.....	......	.....	......
PAWP	.....	......	.....	......
Minute Vent	.....	......	.....	......

Position ....

Time (mins)	0 Event	5 Event	15 Event	30 Event
Time:				
HR	......	......	.....	......
BP	......	......	.....	......
MAP	......	......	.....	......
PAP	......	......	.....	......
MPAP	......	......	.....	......
PCWP				......
RA	......	......	.....	......
Respiratory:				
Rate	......	......	.....	......
VT	......	......	.....	......
PAWP	......	......	.....	......
Minute Vent	......	......	.....	......

Arterial ABG:
Time:
FiO_2

PaO_2	......	......	......	......
$PaCO_2$	......	......	......	......
H	......	......	......	......
SaO_2	......	......	......	......
BE	......	......	......	......

Mixed Venous ABG:

PvO_2	......	......	......	......
$PvCO_2$	......	......	......	......
H	......	......	......	......
SvO_2	......	......	......	......
BE	......	......	......	......

Chest X-Ray Report:

Immediate Post-Op:
....

Post-Op Day 1:
....

Post-Op Day 2:
....

Post-Op Day 3:
....

APPENDIX F

The Study Protocol

1. Potential subjects will be identified by the Patient Educator Nurse to ensure the pre-operative selection criteria are met.
2. The subject will be informed of the study by the Patient Educator Nurse.
3. The name of the interested subject will be forwarded to the researcher. the subject will be approached for an informed consent. Each subject will be made aware that the frequency of turning is in excess of the standard practice of the unit (every two hours). Furthermore, the subject will be advised that there will be pain associated with the frequent turning but an analgesic will be give prior to the study and at the completion of the study. The duration of the study will last approximately two hours.
4. Post-operatively, the subject's flow chart will be reviewed to satisfy the post-operative selection criteria. Additional information as outlined on the data collection sheet will be obtained either before the study protocol or afterwards.
5. Each subject will be give an intravenous analgesic, prior to the initiation of the study. The researcher will collaborate with the bedside nurse to ensure the correct timing of the analgesic.
6. There will be random assignment to the starting

position according to the sequencing schedule.

7. The headrest elevation of 30° will be standardized using a 30° angular ruler. The transducer will be levelled at the phlebostatic axis using a carpenter's level. Cardiac output will be measured when the patient is in the supine position.

8. Initiation of the study will be timed so that it will occur either before routine airway suctioning or half-an-hour after suctioning.

9. A base line arterial and mixed venous blood samples will be drawn in whichever position the subject is found. The mixed venous blood sample will be drawn from the distal port of the pulmonary artery catheter with a discard of three millilitres (ml) of blood, and one ml of mixed venous blood will be obtained anaerobically in a pre-heparinized three mls syringe. The arterial blood sample will be obtained from the arterial line with a discard sample of two mls of blood. The arterial blood sample of one ml will be obtained anaerobically with another pre-heparinized syringe. These two blood samples will be drawn by the researcher simultaneously. The blood will be stored in ice immediately and transported to the laboratory within five minutes. The duration of the subject in this position will be recorded.

10. The subject will begin in one of the three possible starting positions. The lateral position will be maintained by a wedge foam. The subject will be assisted to turn on his or

her side by two persons (the researcher and the research assistant). Changes in vasoactive medications will be recorded on the data collection sheet. If suctioning is required during the study, the subject will be suctioned and the study protocol will resume after a waiting period of 30 minutes. However, no changes in ventilator settings will be made during the study. If these changes are necessary, the subject will be dropped from the study.

11. Once the subject is positioned, there will be a waiting period of 30 minutes before the blood sampling. Two blood samples will be drawn, a mixed venous and an arterial blood samples.
12. Hemodynamic and respiratory parameters will be obtained before the patient is repositioned. Steps 11 to 12 will be repeated until the patient has assumed all three positions.
13. The immediate post-operative and the next morning routine chest X-ray report from the radiologist for presence or absence of atelectasis will be recorded in the data sheet when available.